The prevalence of patellar tendinopathy in elite academy rugby players: a role for measuring ground reaction forces?

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Abstract

Discussions with rugby team doctors and physiotherapists suggest that the prevalence of patellar tendinopathy (PT) in rugby populations is increasing. However, from a review of the published literature patellar tendinopathy is not a condition commonly reported in rugby athletes. PT can have serious consequences for an athlete, which may even be career ending. Some of the characteristic ultrasound features of tendinopathy can be seen prior to an athlete developing symptoms of the disease. This would suggest that players should be regularly examined with ultrasound. However, such an approach is prohibitive because of the cost, both of the equipment and the professional expertise required. Therefore, a screening method, which did not involve the use of expensive equipment and trained personnel, and which could predict the presence of patellar tendon disease, especially when the athlete is asymptomatic, is highly desirable. To date, such a screening method does not exist. The aims of this study were to investigate, in a sample of elite academy rugby players: 1) the prevalence of patellar tendinopathy, 2) the prevalence of asymptomatic patellar tendon disease on ultrasound imaging, 3) whether patellar tendon disease is associated with weight or playing position, 4) whether vertical ground reaction force (VGRF) data from drop-landing tasks can predict the presence of patellar tendon disease and avoid the need for costly ultrasound screening.

A group of academy rugby players from Wales (n=46) received a combination of clinical and ultrasound examination of their patellar tendons. Ultrasound examination was used to establish the presence of a patellar tendon abnormality (PTA). VGRF data were measured during a drop-landing task from a 30 cm platform situated immediately behind a portable force plate. Peak VGRF and loading rate VGRF data were recorded.

Sixteen players (35%) were found to have patellar tendon disease (a PTA) on ultrasound imaging. The prevalence of clinically apparent PT, with associated ultrasound changes, in this cohort was found to be 15.2% (n=7). There was no

difference in either body mass or playing position between those players with patellar tendon disease and those with healthy patellar tendons. VGRF data from a drop-landing task was unable to predict the presence of patellar tendon disease.

The study suggests that the prevalence of patellar tendon disease is higher than previously reported. The study does not support the use of VGRF to predict the presence of patellar tendon disease. Due to the high prevalence of disease, and the potential consequences for those affected, the study recommends further research into prevalence, risk factors and screening for this condition in elite rugby players.

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My grateful thanks go to Prof Gareth Lloyd Jones who carried out the clinical examinations and accompanied me to the regional clubs to collect data.

The two Pauls from the University of South Wales were a helpful source of sound advice especially at times when I doubted myself. Thank you to Paul Gill and Paul Jarvis.

Family are always a vital source of support when a thesis is being written. It was no different for me. Support from my family came in so many ways: meals were given; proof reading undertaken and solace, comfort, motivation and praise given in equal measure. They have my love and gratitude.

Dedication

This thesis is dedicated to rugby football. To a sport that gives a nation its identity and to the players whose skill is the source of so much enjoyment and pleasure. Above all, I dedicate this work to those players whose lives are changed forever due to injury.

Contents

Abstract ii
Acknowledgements iv
Dedication v
List of abbreviations viii
List of figures and charts ix
List of tables x
Chapter 1. Introduction to the Study 1
Chapter 2. Patellar Tendinopathy: a review of the literature 3
2.1 Introduction 3
2.2 Search strategy 3
2.3 The Patellar Tendon 4
2.4 Definition of Patellar Tendinopathy 5
2.5 Pathogenesis and Symptomatology 6
2.6 Diagnosis of Patellar Tendinopathy 10
2.6.1 Clinical Examination 11
2.6.2 Musculoskeletal imaging modalities used in the diagnosis of PT 11
2.7 Epidemiology of Patellar Tendinopathy 13
2.7.1 Prevalence in elite athletes 13
2.7.2 Prevalence in non-elite athletes 15
2.7.3 Prevalence in Rugby 15
2.8 Impact of Patellar Tendinopathy 17
2.9 Risk Factors for the development of Patellar Tendinopathy 19
2.9.1 PTA 19
2.9.2 Anthropometric risk factors 20
2.9.3 Playing position 22
2.9.4 Ground Reaction Forces 23
2.9.5 Previous injuries 25
2.9.6 Genetics 25
2.10 Conclusions 26
Chapter 3: Methodology 28
3.1 Introduction 28
3.2 Aims and Objectives of the study 28
3.3 Study Design 29
3.4 Ethical Approval 30
3.5 Sample 31
3.6 Inclusion / Exclusion Criteria 33
3.7 Recruitment Strategy 34
3.8 Baseline demographic data 35
3.9 Baseline symptom score (for use as a clinical management tool if PT or PTA was diagnosed) 36
3.10 Clinical Examination 37
3.11 Ultrasound imaging 40
3.12 Drop-Landing Task 44
3.13 Statistical Analysis 47
Chapter 4: Results 49

4.1 Introduction 49
4.2 Sample 49
4.3 Prevalence 50
4.4 Body Mass 51
4.5 Playing position 52
4.6 Vertical Ground Reaction Force (VGRF) 54
Chapter 5: Discussion 58
5.1 Introduction 58
5.2 Sample 58
5.3 Prevalence 58
5.4 Body mass 64
5.5 Playing position 66
5.6 Vertical Ground Reaction Force 66
Chapter 6: Conclusions 70
Limitations 72
Recommendations for practice and research 73
References 76
Appendix 1. Ethical approval 87
Appendix 2. Study invitation letter to regional rugby academies 88
Appendix 3. Participant Information Sheet 89
Appendix 4. Consent form 93
Appendix 5. VISA-P questionnaire 95
Appendix 6. Experience of clinician conducting clinical examination and undertaking ultrasound scans of patellar tendons 97
Appendix 7. Pro-forma for data collection: clinical examination 99
Appendix 8. Pro-forma for data collection: ultrasound examination 100

List of abbreviations

BMI	body mass index
CD-US	colour-doppler ultrasound
CT	computerised tomography
GS-US	grey scale ultrasound
IC	initial foot ground contact
IRB	international rugby board
LR VGRF	loading rate vertical ground reaction force
MRI	magnetic resonance imaging
PT	patellar tendinopathy
PTA	patellar tendon abnormality
RCT	randomised control trial
RWC	Rugby World Cup
tVGRF	time to peak vertical ground reaction force
US	ultrasound
VGRF	vertical ground reaction force
VISA-P	Victorian Institute Sport Assessment for PT

List of figures and charts

Figure 2.1: Anatomy of the Knee 4

Figure 2.2: Theory of pathology continuum. 7

Figure 2.3: Prevalence of of jumper's knee in elite athletes 14

Figure 3.1: Study participant undergoing lunges 38

Figure 3.2: Examiner testing for tenderness over the insertion of the patellar tendon 39

Figure 3.3: Position of the participant for ultrasound scanning of the patellar tendon 41

Figure 3.4: Orientation of the ultrasound transducer to visualise the tendon in the transverse plane 42

Figure 3.5: Orientation of the ultrasound transducer to visualise the tendon in the longitudinal plane 42

Figure 3.6: Demonstrating set up of equipment for drop-landing task 45

Figure 3.7: Graphical representation of acquired ground reaction force-time curve for drop landing task 46

Chart 4.1: Distribution of results according to disease group 50

Chart 4.2: Body mass according to disease group 51

Chart 4.3: Distribution of results in forwards 53

Chart 4.4: Distribution of results in backs 53

Chart 4.5: Peak VGRF according to disease group 54

Chart 4.6: LR VGRF according to disease group 55

List of tables

Table 4.1: Comparison of body mass between control and diseased groups 52

Table 4.2: Descriptive statistics for VGRF and LR VGRF 56

Table 4.3: Logistic regression model predicting likelihood to suffer diseased patellar tendon 57

Table 5.1: Comparison of demographic information with Durcan et al. (2013) 59

Table 5.2: Comparison of results with Durcan et al. (2013) 60

Table 5.3: Comparison of results with senior regional cohort 63

Chapter 1. Introduction to the Study

This study is about patellar tendinopathy (PT) in rugby players. It is a condition that is thought to be increasingly common and difficult to diagnose outside a clinical environment. It can be present prior to an athlete experiencing symptoms of the disease. Once established, it can have significant consequences for the player and, in a worse case scenario, patellar tendinopathy can end an athlete's playing career. Diagnosis of PT is made by clinical and ultrasound examination of the patellar tendon (the gold standard) which is not often routinely available. This means that PT can deteriorate undetected, which makes subsequent treatment and management more prolonged with possible devastating consequences for the athlete. In this study, an attempt is made to determine the prevalence of PT in a sample of age-grade rugby players attached to academies; and to investigate whether it is possible to predict the presence of PT by using a test that is simpler to administer than the gold standard.

Patellar tendinopathy is a common sporting overuse injury. The most widely referenced study on PT prevalence is by Lian et al. (2005) who reported that the overall prevalence of patellar tendinopathy in elite sports (not including rugby) is approximately 14%. PT is more commonly seen in sports that are characterised by high demands on leg extensor speed and power (Visnes et al., 2012). This includes sports that involve high frequency jumping activities or those which involve quick changes in direction (cutting). It is thought that amongst other factors, this is due to high patellar tendon load[1] (Bisseling et al., 2007; Cook & Purdam, 2009; Scott et al., 2013). Thus, the more commonly researched sports for this condition include volleyball, netball, basketball and soccer. Rugby, which has all of the above characteristics, is devoid of studies into patellar tendinopathy and therefore the prevalence of patellar tendinopathy is unknown in rugby players. Chalmers (2002) argues that from a "national sports organization" perspective, injury prevention research should be targeted towards injuries that are common, that prevent elite

[1] The sum of all the forces and moments acting on a body [the patellar tendon](Kent, 1994)
[2] Coombes et al. (2009); Lewis (2010); Malliaras & Cook (2011); Campbell et al. (2013).

athletes from competing and earning revenue for their sport, and those that are the greatest barriers to personal achievement. Several oral reports from sports doctors working within rugby union in Wales claim that the prevalence of patellar tendinopathy is increasing (personal communication Jones, 2010; Ridgewell, 2012). However, if one takes the view that "the pleural of anecdote is not data" (Frank Kotsinis), a study in to the prevalence of PT in rugby is warranted.

Despite rugby being a popular, professional sport worldwide, there are few studies that have established either the prevalence of patellar tendinopathy in rugby players or a mechanism for predicting its presence. This study attempts to address these gaps in understanding. It is expected that the prevalence of patellar tendinopathy in rugby athletes would be similar to that of the sports which have been reported on, because rugby involves similar movement patterns.

In order to undertake a study of PT in rugby players, the pathogenesis of the condition and its effect on athletes needs to be understood. The literature review that follows addresses the pathogenesis and symptomatology of patellar tendinopathy and examines the insidious nature of symptom development. Studies that have examined the condition are reported on. Current practice in relation to diagnosis is discussed and the impact of PT is also explored. The conclusions inform both the study questions and design.

Chapter 2. Patellar Tendinopathy: a review of the literature

2.1 Introduction

As this study is about patellar tendinopathy and its potential impact on the career of elite rugby players, this chapter begins with an analysis of the pathological changes that can occur as a result of overuse. It then goes on to provide an analysis of what is currently understood about both the prevalence and aetiology of PT and its associated risk factors.

2.2 Search strategy

A comprehensive literature review of tendinopathy, with specific emphasis on patellar tendinopathy was undertaken. The search strategy involved a free text search across the MEDLINE database. Academic search engines (such as Google Scholar) were key because no one database contained all the relevant journals. Areas such as clinical medicine, biomechanics, sport injury and screening etc. are not all contained in one database. Whether searching MEDLINE or search engines, the same search terms were used. Initial search strings were expanded as different evidence was accumulated. For example, an initial search on tendinopathy expanded subsequently to include 'degenerative' and 'inflammatory' in the string. There were no time parameters in the search. Only publications that were in English were included. Traditional search engines were used to locate grey literature such as technical reports from working or research groups. Key search terms included, but not limited to, 'tendinopathy', 'tendinopathy rugby', 'patellar tendinitis', 'jumper's knee', 'patellar tendinopathy' and 'patellar tendinopathy rugby'. Searches were repeated throughout the course of the dissertation to alert the researcher to new publications. This proved to be crucial as a study very similar to the one reported here was published in 2013. Likewise a paper, which suggested that some of the damage seen in tendinopathy might be mediated through inflammatory processes, was also

published in 2013. Social networks were used to alert the researcher to new publications relating to tendinopathy.

2.3 The Patellar Tendon

The patellar tendon is an extension of the quadriceps femoris tendon. It extends from the inferior pole of the patella and inserts on to the tibial tuberosity (figure 2.1).

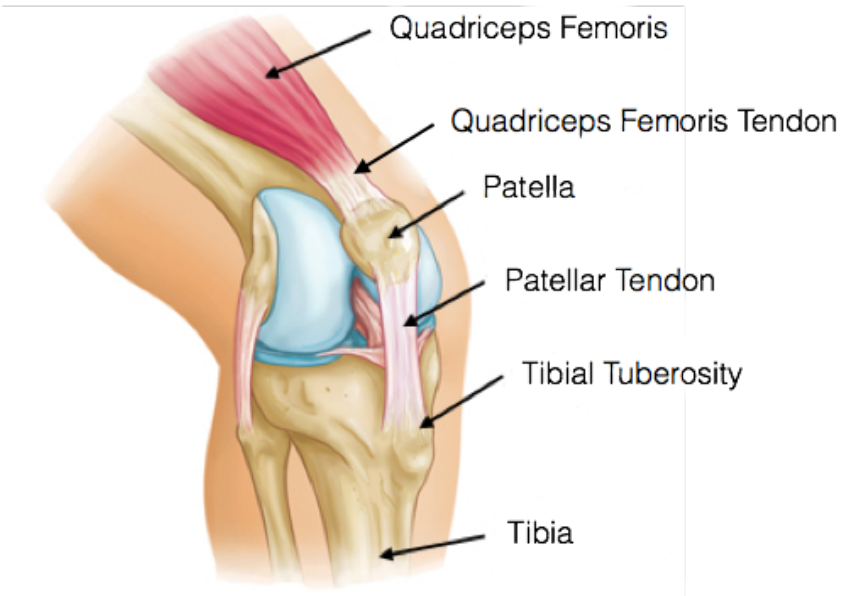


Figure 2.1: Anatomy of the Knee. Source: Orthoinfo, American Academy of Orthopaedic Surgeons (n.d.)

The patellar tendon, along with the quadriceps muscle and tendon, the patella and the retinaculum (the strong band of connective tissue that holds the patella in place), make up the extensor mechanism of the knee, which enables the leg to be straightened.

2.4 Definition of Patellar Tendinopathy

Patellar tendinopathy is a clinical condition characterised by activity related, anterior knee pain (Brukner et al., 2007). This pain is accompanied by tenderness to palpation over the patellar tendon or its attachments, particularly over the inferior pole of the patella. It is one of the most common of the chronic tendinopathies (Maffulli et al., 2003). The term "patellar tendinopathy" has become the preferred term for the conditions previously described as "jumper's knee" or "patellar tendinitis" (Khan et al., 1999). This change in nomenclature reflects the change in understanding of the pathogenesis of the condition in the 1990s. The consensus at that time was that tendinopathy was no longer thought to be inflammatory in nature, but degenerative. Thus, the term "tendinitis" is a misnomer and this has been the prevailing understanding of the condition for circa 20 years. In 2009, Cook and Purdam reported on a critical review of histopathological and imaging studies into tendinopathy. Their conclusions supported the prevailing opinion that tendinopathy is degenerative in nature. Recently, however, Rees et al. (2013) queried whether:

1. At least some of, the damage seen in tendinopathy was mediated by elements of the inflammatory process and
2. The prevailing understanding needed to be challenged?

They felt that to refer to tendinopathy as degenerative only was an oversimplification. This premise was based upon a number of small-scale studies that demonstrated microscopic evidence of inflammation being present in tendinopathy (Schubert et al., 2005). Also in 2013, the International Scientific Tendinopathy Symposium consensus statement on exercise related tendinopathy was published (Scott et al., 2013). Two of the authors of Rees et al., (2013) were also contributors to the consensus statement in which, it was accepted that, "structural degeneration of the load-bearing matrix [of the tendon] is a key feature, with an absence or minimal presence of inflammatory cells". Nevertheless, Rees et al. (2013) argue that the contribution of inflammatory processes as a key component of tendon damage needs to be appreciated. The challenges posited by Rees et al. (2013) to the current understanding of the condition must be given consideration (particularly when successful treatment is difficult). However, until further appropriate research is

available, for the purposes of this study, tendinopathy is regarded as mainly degenerative in nature.

2.5 Pathogenesis and Symptomatology

Tendinopathy, viewed as a degenerative process, is most often described as a mechanism of dysrepair (failed healing) in response to chronic overload of the tendon. Both extrinsic factors (e.g. sport surface, load) and intrinsic factors (e.g. biological age, sex, genes, biomechanics) have been implicated in predisposing to injury but according to Cook & Khan (2008), who reviewed existing studies, the pathoaetiology is not clear. However, there does seem to be a consensus that load is a critical factor (Bisseling et al., 2007; Cook & Purdam, 2009; Scott et al., 2013). Given this common understanding, Cook & Purdam (2009) examined the existing evidence base for load as a factor in tendinopathy. In an attempt to understand this relationship, 83 papers were reviewed (of which they had contributed to the authorship of n=12). The studies included in their paper fall into three broad categories:

1. The histopathological changes associated with tendinopathy
2. The outcome from various treatments for tendinopathy
3. The way in which the disease progresses over time (including periods of rest or reduction of load).

The conclusion from the analysis of the included studies is that tendon pathology might be conceptualised as a continuum, which Cook & Purdam (2009) represent as a model (figure 2.2). The model is described and critiqued in some detail here because it captures existing theories about the way in which the disease progresses forwards and backwards along the pathology continuum.

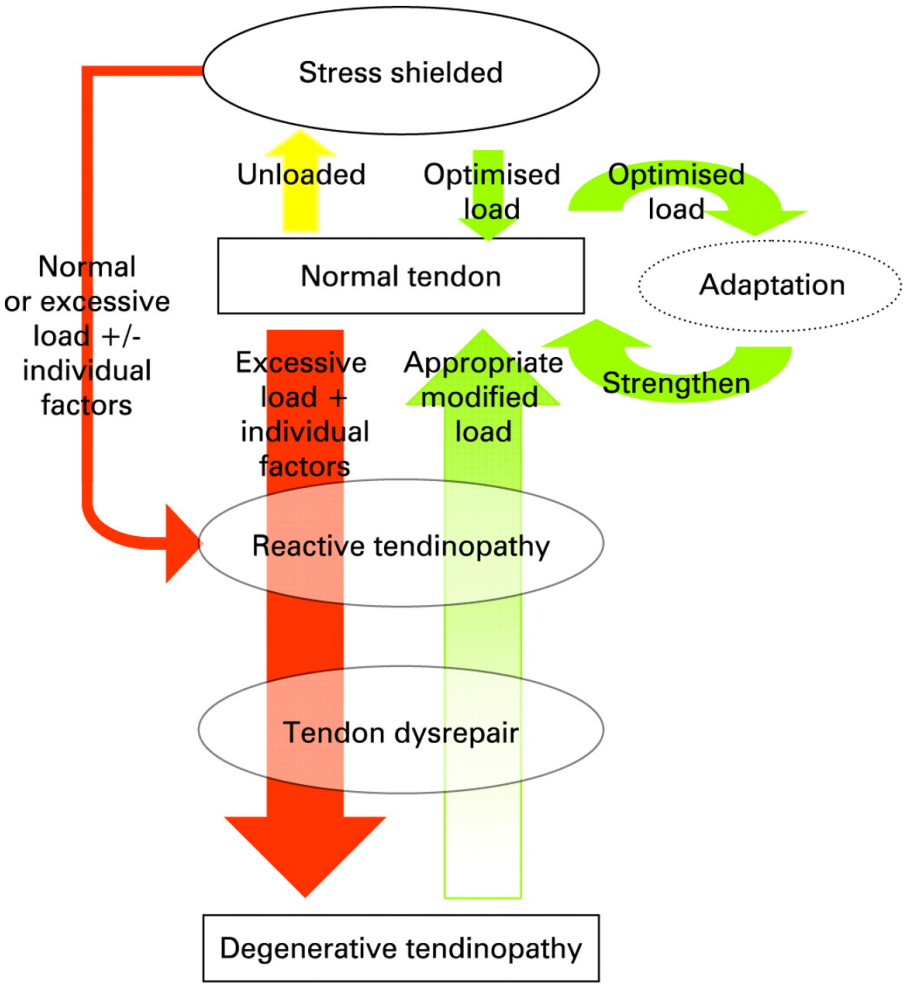


Figure 2.2: Theory of pathology continuum. Reproduced from Cook & Purdam (2009)

A model is an abstraction of reality, the usefulness of which is its ability to be used as a comparison for reality. This model is described in detail below. Although citing load as the main catalyst for degenerative tendinopathy, Cook and Purdam (2009) acknowledge that the pathoaetiology is "almost certainly" modulated by a range of other factors. The model is frequently cited (see footnote)[2], including in the International Scientific Tendinopathy Symposium consensus statement (Scott et al., 2013).

[2] Coombes et al. (2009); Lewis (2010); Malliaras & Cook (2011); Campbell et al. (2013).

Cook and Purdam's (2009) model describes three stages along this pathology continuum: reactive tendinopathy, tendon dysrepair and degenerative tendinopathy. The addition or removing of load is thought to be the primary stimulus that drives the health of the tendon forward and back along the continuum.

Reactive Tendinopathy

Reactive tendinopathy is the first stage along the continuum, a result of acute tensile or compressive overload of the tendon e.g. an academy rugby player who dramatically increases the amount of training sessions performed per week. The tendon adapts by becoming thicker, this reduces stress by dissipating the force over a larger surface area. This area of thickening can be seen on ultrasound scanning of the tendon (as will be discussed). The athlete in question may exhibit patellar tendon swelling and pain. Cook and Purdam described reactive tendinopathy as a short-term adaptation to overload. It is thought that tendons in this stage of the continuum can revert back to normal if a sufficient reduction of load occurs or there is sufficient rest between loading sessions (Cook & Purdam, 2009).

Tendon Dysrepair

The second stage along the continuum is termed tendon dysrepair. It is thought to be a failed healing response to reactive tendinopathy. This can occur when there is insufficient removal of load i.e. continuing to train at high intensity (volume or frequency) with inadequate rest periods. This would mean that the pattern of training described in the reactive tendinopathy stage persists. In similar fashion to academy rugby players, Reeser et al. (2006) argued that in volleyball players, the risk of developing patellar tendinopathy is higher at the time when players are promoted from junior level to senior level. The abrupt move from a relatively safe training environment, which might involve practice two or three days a week to an elite sport environment; which has a frequent, often daily, structured programme of activity (training or playing); results in a high risk of developing patellar tendinopathy.

Tendon dysrepair is characterised by a marked increase in protein production (collagen and proteoglycans). The increased proteoglycans result in separation of the collagen fascicles and disorganisation of the usually well-structured matrix. The relevance of this will become apparent later when describing the gold standard for diagnosing patellar tendinopathy, which involves ultrasound scanning of the tendons that, at this stage, will show the areas of collagen disorganisation as focal areas of hypoechogenicity[3]. Cook and Purdam (2009) acknowledge that this stage of pathology can be difficult to distinguish clinically.

Degenerative Tendinopathy

The most extreme end of the continuum is termed degenerative tendinopathy. It is a progression of the changes seen in the previous stage. The areas of disorganisation of the matrix are larger. This is reflected as larger areas of hypoechogenicity on ultrasound imaging. It is thought that the potential for reversibility from this stage is limited. If the degeneration is significant enough, the tendon has the potential to rupture. Where there is degenerative tendinopathy, the athlete would require significant treatment and the damage might even be so great as to be career ending.

The development of patellar tendon disease can be insidious because the athlete may not experience symptoms despite degenerative changes being present, that is, a patellar tendon abnormality (PTA) can be seen on ultrasound imaging in a patient who is asymptomatic (as will be discussed). Thus, an athlete can have an abnormal patellar tendon but not experience any symptoms. Asymptomatic PTA can result in the athlete continuing to overload the tendon, which, in Cook & Purdam's model, would drive the health of the tendon along the continuum toward degenerative tendinopathy.

Cook & Purdam (2009) state that the suggestions of the model are derived from analyzing cross-sectional studies and "supported by findings in animal models".

[3] a lesser ability to produce echos compared with neighbouring structures. Appears as varying shades of dark grey or black on ultrasound (Silkowski, 2010)

They acknowledge that there is limited evidence available from clinical studies. This might be what the authors are referring to when they state, “the integrity of the model will only be as good as its capacity to withstand additional research”. Furthermore, a critique, which is a polemic (Robinson, 2010) and not an empirical piece of work, challenges the influence of load as a catalyst. It criticises the “presumption” of overload as the key initiating factor and argues that the continuum theory does not explain the “large population who develop tendon pain without overload / overuse”. The model, which includes both manifestation and management, could be made even more useful if terms associated with “load” and “individual factors” were more clearly defined. For example, “normal”, “excessive”, “appropriate” and “optimized” are terms which lack precision. Overcoming this may increase the clinical utility of the model.

Despite these limitations, Cook & Purdam’s model is frequently cited (as discussed) as being useful clinically because it appears to capture possible disease trajectories. This gives it a measure of face and possibly content validity. It also has utility in communicating, to an athlete, the risk of disease progression in the absence of any modification in training regimens. In order for this to be successful, however, there is a requirement to ensure that an accurate diagnosis of tendinopathy takes place. The next section in the literature review addresses this issue.

2.6 Diagnosis of Patellar Tendinopathy

The diagnosis of PT is based primarily on a history of characteristic symptoms (activity related, anterior knee pain) and positive clinical examination findings (Cook et al., 2001). The diagnosis is then often supported with consistent musculoskeletal imaging findings, that is a patellar tendon abnormality (PTA). Each of these diagnostic processes is discussed and reviewed below.

2.6.1 Clinical Examination

The examination for PT involves palpation of the patellar tendon and it's bony attachments. As the patellar tendon lies immediately beneath the skin, it can be easily palpated. Classically, athletes with patellar tendinopathy will experience pain on palpation of the inferior pole of the patella and or the proximal patellar tendon (Brukner et al., 2007). The examination also includes asking the patient to perform functional movements such as squatting, lunging or hopping to load the patellar tendon (Brukner et al., 2007; Cook & Purdam, 2009). Such movements aim to reproduce the pain of patellar tendinopathy.

2.6.2 Musculoskeletal imaging modalities used in the diagnosis of PT

The clinical diagnosis of PT is usually confirmed with musculoskeletal imaging. Both ultrasound imaging and Magnetic Resonance Imaging (MRI) are commonly used in confirming clinically diagnosed PT (Warden et al., 2007). These have advantages over other diagnostic modalities such as Computed Tomography (CT). For example, unlike CT scans, ultrasound and MRI modalities do not expose patients to potentially harmful radiation. Warden et al., (2007) investigated the comparative accuracies of US and MRI in detecting PT by comparing a cohort of 30 patients with clinically diagnosed patellar tendinopathy with 33 matched healthy controls (i.e. without PT). They found that ultrasound imaging was more accurate than MRI in detecting clinically diagnosed patellar tendinopathy (83% vs 70%, P0.04). Grey-scale ultrasound (GS-US) was found to be the most sensitive (i.e. The proportion of people with symptoms of PT who have a positive test result) at detecting patellar tendinopathy (sensitivity of 87% vs 57% for MRI, P= 0.01). The specificity (i.e. The proportion of people without symptoms of PT who had a negative test result) for both US and MRI modalities was 82%.

The authors also used colour doppler ultrasound (CD-US), which is used to examine the knee in a different way from grey-scale ultrasound. CD-US reveals blood flow. Neoneurovascularisation (the development of new blood vessels and accompanying nerves) would be indicative of the healing process, which only occurs as a

consequence of damaged tissue. In a longitudinal study over five months in volleyball players in Australia, Cook et al. (2005) found that persistent vascularity on CD-US was associated with increased pain scores ($p=0.043$). It is thought that the neoneurovascularisation may be responsible for, or at least contribute to, the pain experienced in PT (Alfredson et al., 2003). This provides the basis for one of the treatment modalities for PT i.e. sclerosing therapy, which eradicates newly developed nerve fibers.

In the Warden et al. (2007) study, CD-US had good accuracy as a result of its ability to confirm when PT was diagnosed on clinical examination (sensitivity) and its very good ability to confirm when clinical examination was negative for PT (specificity). Warden et al. conclude that GS-US combined with CD-US may represent the imaging approach of choice. GS-US should be used given its good sensitivity (87%), while CD-US enhances the diagnostic ability because of its high positive predictive value (proportion of those who test positive, who actually have symptoms of PT, 91%). This makes GS-US coupled with CD-US the gold standard, which explains why this imaging modality has been used in the majority of patellar tendon studies.

Ultrasound imaging has further benefits over other radiological investigations, it is both time and cost efficient when compared to CT and MRI, especially when scanning large numbers of individuals such as those involved in team sports. However, providing ultrasound investigation for all athletes, especially those involved in team sports, would be prohibitive. It is dependent on having the expertise of an ultrasonographer and possibly a radiologist for reporting. In addition, the necessary equipment is expensive. Therefore, a method of screening for PT, which in the first instance would not require the expertise of medically qualified professionals, could be very beneficial. It would allow comprehensive coverage of *at risk* athletes. Those with any indication of PT would be followed up with gold standard investigation i.e. GS-US and CD-US.

There would need to be strong justification for comprehensive screening of all professional (elite or sub-elite) rugby players even with an easy to perform predictive test. The justification is set out below:

1. Degenerative changes in the tendon (PTA) can be identified before an athlete experiences symptoms thus avoiding further damage to the tendon from continued overuse (as discussed above).
2. The perceived scale of the problem (epidemiology).
3. The condition can be career limiting (impact).

Epidemiology and impact are dealt with below.

2.7 Epidemiology of Patellar Tendinopathy

2.7.1 Prevalence in elite athletes

Prevalence is defined as the proportion of a population that suffer from a particular disease at a particular moment in time (Dancey et al., 2012). It has been reported that patellar tendinopathy has an estimated overall prevalence of approximately 14% in general elite sporting populations (Lian et al., 2005; figure 2.3), with an additional 8% reporting previous symptoms of patellar tendinopathy. In this cross-sectional designed study, Lian et al. (2005) used a combination of injury questionnaire and clinical examination in elite Norwegian athletes. The estimated prevalence corresponds to one in five athletes being affected during their career.

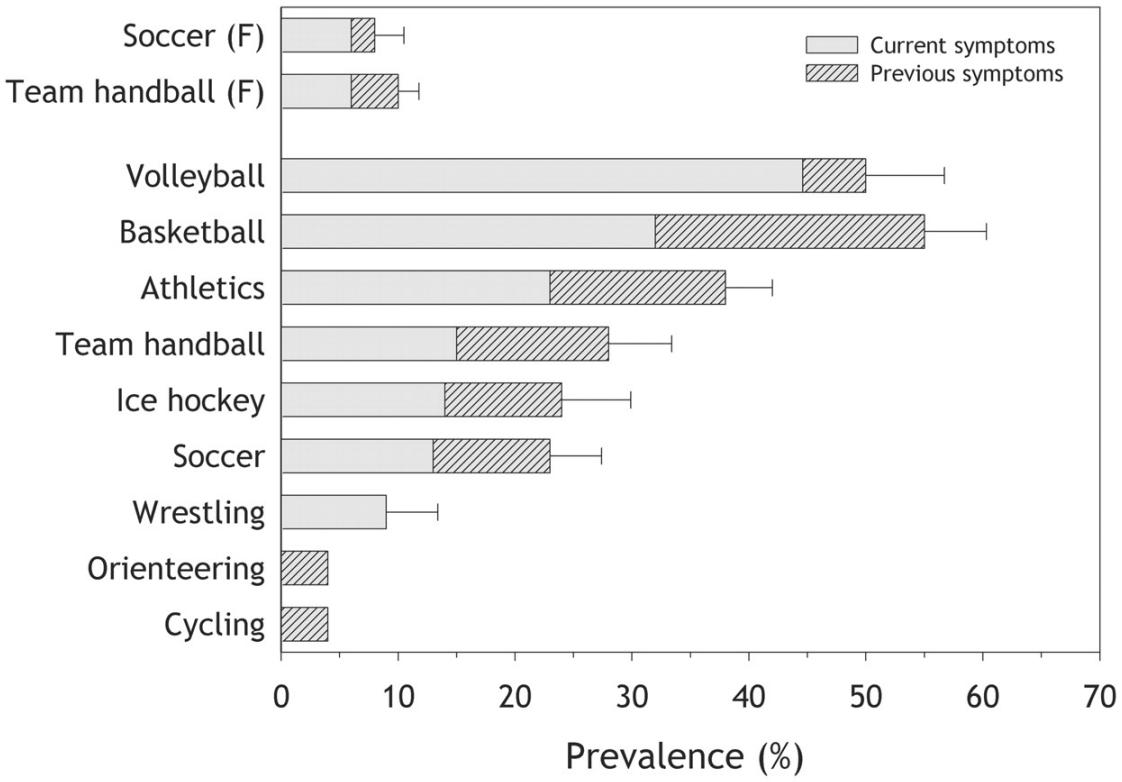


Figure 2.3: Prevalence (%) of current (gray bars) and previous (hatched bars) symptoms of jumper's knee. The results for female athletes (F) are shown in the 2 upper bars; the rest of the results are for male athletes. Error bars denote SE (Lian et al. 2005).

The high prevalence of patellar tendinopathy in elite male volleyball athletes is consistent across several studies with rates of approximately 40-50% (Ferretti et al., 1984; Ferretti et al., 1990; Lian et al., 1996). It is thought that the sports with the highest prevalence of patellar tendinopathy are those that are characterised by high demands on speed and power of the leg extensors (Lian et al., 2005; Visnes et al., 2012). They are typically sports, like rugby, that involve jumping, sprinting, and cutting. This explains why the sports with the highest reported prevalence are basketball, volleyball, athletics and soccer. More recent studies and systematic reviews rely on the results of Lian et al. (2005) for prevalence estimations.

2.7.2 Prevalence in non-elite athletes

A cross-sectional survey performed by Zwerver et al. (2011), reported that the overall prevalence of "jumper's knee" in non-elite athletes from different sports (basketball, volleyball, handball, korfball, soccer, field hockey and track and field) was 8.5%. The sport with the highest prevalence was again found to be volleyball (prevalence of 14.4%). This is the only study of non-elite athletes reporting prevalence across different sports (rugby was not included in this study). There is increasing penetration of rugby in the general population through programmes such as Rugby Allstars, which is the Welsh Rugby Union scheme that begins at age three. This means that the population of non-elite rugby players is increasing. There are no reported prevalence estimates for such players. Given the findings by Zwerver et al. (2011), this will need to be given due consideration.

2.7.3 Prevalence in Rugby

Rugby is devoid of studies into patellar tendinopathy and as such, the prevalence of patellar tendinopathy in rugby is not known. Since rugby became a fully professional sport in 1995, there have been few large scale epidemiological studies concerning injuries. The largest injury surveillance data for rugby injuries comes from the *Epidemiology of injuries in English professional rugby union* studies (Brooks et al., 2005a; Brooks et al., 2005b)). The studies report only three incidences of PT in 546 players over two years. This is much lower than the prevalence estimations of PT in other sports, which involve similar skills to rugby. The explanation for this may lie in Brooks et al. (2005a, p. 757) definition of an injury, which is:

> *"any injury that prevents a player from taking a full part in all training and match play activities typically planned for that day for a period of greater than 24 hours from midnight at the end of the day the injury was sustained."*

This definition has its basis in acute injuries only i.e. those which occur in the match or training environment and lead to time off from training or competition. However,

from an understanding of the aetiology of the condition, using such a definition may well have lead to instances of PT being missed because of its insidious nature. In turn, this may result in players not missing training or competition in that 24 hour window (for reasons that have already been described). This is also true of epidemiology studies into rugby injuries from other countries (Bird et al., 1998; Bathgate et al., 2002).

The International Rugby Board (IRB), the governing board for professional rugby, started collecting injury surveillance data at the Rugby World Cup (RWC) in 1995. They have done this for each of the rugby world cups since. However, only the injury surveillance data collected from the 2007 (Fuller et al., 2008) and 2011 (Fuller et al., 2012) rugby world cups were consistent with the protocol of the International Consensus Statement for Epidemiological Studies in Rugby (Fuller et al., 2007). Thus, the pre-2007 data is not reliable and therefore not comparable with post-2007 data. This means that data exists for two Rugby World Cups only. However, the consensus statement for epidemiological studies in rugby has not addressed the problem in relation to Brooks et al. studies with regards to chronic overuse injury. The consensus statement (Fuller et al., 2007, p. 329) states that an injury is defined as:

> *"Any physical complaint, which was caused by a transfer of energy that exceeded the body's ability to maintain its structural and/or functional integrity, that was sustained by a player during a rugby match or rugby training, irrespective of the need for medical attention or time-loss from rugby activities. An injury that results in a player receiving medical attention is referred to as a 'medical-attention' injury and an injury that results in a player being unable to take a full part in future rugby training or match play as a 'time-loss' injury."*

Once again, using this definition, chronic overuse injuries might be missed and this may well be obfuscating the actual prevalence. This is because the condition may be characterised by its insidious onset, which may not be connected to a particular match or training session. It can also cause confusion because one player could fall into both categories. In addition, the problem is compounded because of coding issues. As far as can be ascertained from the report, the coders may have classified

PT severally as either “knee injuries” or “tendon rupture / tendinopathy / bursitis” (not specifying joint affected). The reporting of injury rates in this way is advocated in the consensus statement on data collection procedures for studies of injuries in rugby union (Fuller et al., 2007). However, as these codes may cover a wide variety of pathologies, an accurate estimate to the prevalence of PT cannot be made. In order to obtain clarity on coding issues, contact was attempted with the authors of Fuller et al. (2012). However, no reply to our communication was received.

Furthermore, the data from the RWC surveillance studies report incidence and not prevalence. Incidence is defined as the rate at which injury occurs “during a given period in a specified population” (Last, 2000). As patellar tendinopathy is a chronic condition, incidence rates over a relatively short period (e.g. 7 weeks in a rugby world cup) must be interpreted with caution. Bahr (2009) recognises that the current method of surveillance data is inadequate and highlights the need for new approaches to quantify overuse injuries in studies.

Increasingly, conversations with rugby sport doctors (and from my own practice) suggest that PT is becoming one of the most common chronic injuries presenting to health professionals working with rugby athletes. The first aim of this study is to investigate the prevalence of patellar tendinopathy in rugby union athletes. It was hypothesised that the prevalence of PT in rugby athletes would be similar to, or greater than comparable sports i.e. approximately 14%. When this (Giles) study began in 2011, useful prevalence data were absent, which is one of the main reasons why the study was undertaken. However, In July 2013, Durcan et al. (2013) reported on a study of elite academy rugby players in Ireland and reported a prevalence of PT of 9.6%. The authors assert that this finding is concerning given that PT is not historically associated with rugby. The significance of this finding is discussed in relation to both the findings of this thesis and the prevalence reported in the literature on sports, other than rugby, which are characterised by high demands on leg extensor speed and power (as previously discussed).

2.8 Impact of Patellar Tendinopathy

Athletes with diagnosed patellar tendinopathy may require a prolonged period of time away from competition (months to years) while other athletes may never return to their full pre-injury level of competition or be forced to retire. For example, Kettunen et al. (2002) used the Turku Sports Medicine research unit outpatient department database to identify athletes with and without "jumper's knee". In the fifteen-year follow up, it was discovered that 53% (n=9) of the jumpers knee group had reported giving up their sporting career due to knee problems. This compares with only 7% of those without jumper's knee who claimed to have retired from their sport due to the knee problem. Although involving small numbers of participants, this is the only study located which reports the long-term sequelae of patellar tendinopathy.

In part, the impact of PT is inextricably linked to the requirements for managing the condition. Current treatment modalities for patellar tendinopathy include rest, analgesia, eccentric loading exercises, extracorporeal shock-wave therapy, sclerosing or high volume injections, treatment with blood or blood products (to improve tendon healing), and surgery (Khan et al., 1998; Zwerver, 2008; Rees et al., 2009; Scott et al., 2013). However, the most common treatment for PT is conservative (i.e. without surgical intervention). This entails a rehabilitation programme involving eccentric exercise and correction of biomechanical risk factors (as will be discussed) with the latter not yet subjected to rigorous trials. Even successful treatment may require prolonged absences from playing, with Cook et al. (1997, p. 333) reporting that among 100 jumper's knee patients:

> *"67% of these participants could not play or practice for more than four weeks … 34% [of subjects] were unable to play for more than six months, with 19% sidelined for more than 12 months".*

What is clear is that once acquired, patellar tendinopathy can be difficult to treat (Rees et al., 2013) and has serious consequences for both the professional athlete and the individual who plays sport for recreational purposes.

It follows that the best approach to managing patellar tendinopathy is to avoid its development. Van der Worp et al. (2011) argues that because the successful treatment of PT remains challenging, prevention is of the utmost importance and Finch (2006) notes that knowledge of risk factors is essential in developing preventative measures. Two strategies are relevant. The first is to identify risk factors and avoid them; the second is to screen athletes (at least the athletes most at risk) regularly in order to identify patellar tendon abnormalities as early as possible and implement measures to prevent progression. This is particularly important, as a PTA (the characteristic ultrasound changes associated with patellar tendinopathy), may be present prior to the athlete experiencing symptoms (Cook et al., 2000; Fredberg & Bolvig, 2002; Comin et al., 2013) and is discussed in the section that follows.

2.9 Risk Factors for the development of Patellar Tendinopathy

2.9.1 PTA

A Patellar Tendon Abnormality is a patellar tendon ultrasonographic abnormality. It is defined as either a hypoechoic region (an area less able to produce echos compared with neighbouring structures) seen in both the longitudinal and transverse scans of the tendon or a fusiform swelling of the tendon without hypoechoic areas (Cook et al., 2000). PTA may be seen in athletes who are asymptomatic. The reported prevalence of asymptomatic PTA is variable, ranging from 22-32% (Lian et al., 1996; Cook et al., 2000, Fredberg & Bolvig, 2002; Gisslèn et al., 2005; Malliaras et al., 2006). The significance of this remains uncertain. Studies of PTA seen in asymptomatic individuals have shown that there are three possible trajectories for untreated PTA: in serial scans PTA may remain unchanged: the PTA improves: or the PTA deteriorates (Edwards et al., 2010). Qualitative and quantitative analysis of baseline ultrasound images of PTA has been unable to predict which athletes with asymptomatic PTA go on to develop symptoms (Cook et al., 2000). Currently there is no evidence that predicts which athletes with PTA go on to develop symptomatic patellar tendinopathy.

Despite this, several papers have suggested that PTA is a risk factor for developing symptomatic patellar tendinopathy. For those individuals who have PTA, the increased risk of going on to develop symptomatic patellar tendinopathy, is reported as 17% in elite soccer players compared with controls (Fredburg et al., 2002). For elite junior basketball players, Cook et al. (2000) reported a four-fold increase when compared to controls with no PTA. In ballet dancers, the presence of a PTA (on Achilles and patellar tendons) was found to be weakly predictive (p=0.0381) for future disabling symptoms of tendon disease (Comin et al., 2013). Based on a review of a number of empirical studies, Edwards et al. (2010), argued that individuals with a PTA demonstrable on ultrasound imaging, are less able to adapt patellar tendon load to stresses such as muscle fatigue and are therefore at increased risk of developing symptomatic patellar tendinopathy. Cook and Purdam (2009) argue that the loss of tendon structure (which leads to the PTA) results in a tendon that is less capable of sustaining repeated tensile load.

Furthermore, Edwards et al. (2010) argue that, "It remains unknown, however, whether the presence of PTA in asymptomatic athletes affects their landing technique or whether their landing technique might predispose them to developing a PTA and/or patellar tendinopathy". It is important that this potential link is investigated but it outside the scope of this thesis and should be the subject of a follow up study.

2.9.2 Anthropometric risk factors

A major contribution to the literature on risk factors for PT has been provided by Van der Worp et al. (2011) who undertook the only systematic review, which could be located, into risk factors for patellar tendinopathy. As part of the systematic review, 683 relevant papers; which met the inclusion criteria; were located after the initial search. Following a sift of duplicates, 478 papers survived. The inclusion criteria required that studies were empirical research that investigated factors associated with PT. Each study required an experimental group and group of controls. Only ten of the 478 studies met this inclusion criteria and were included in their systematic review.

It is noteworthy that only ten of the original 683 studies survived the quality testing for level of evidence. It is of note that no other systematic reviews and no randomised controlled trials (RCT) were found. In medicine, the RCT is placed near the top of the hierarchy of evidence. Most systematic reviews of clinical conditions would only include evidence garnered from an RCT. In the absence of RCTs, cohort studies and case-control studies produce results in the next level of the hierarchy of evidence. Out of the ten studies that were included, there were six cross-sectional, three case-control studies and one prospective cohort study. One of the studies reported had two distinct parts producing two sets of results making the number of studies that were included 11. The attrition rate from those papers that had the potential to be included in the systematic review was 98.5% or 97.9% depending on whether the starting figure is 683 or 478.

Out of the 11 studies included in the review, eight investigated whether weight is a risk factor for developing PT. This is because theoretically a higher body mass leads to higher loading of the patellar tendon. Three of these eight studies showed an association between weight (or mass) and PT (Lian et al., 2003 who studied males only, P=0.05; Malliaras et al., 2007, $p<0.01$; Crossley et al., 2007 whose studies included males and females, $p<0.02$). The first two of these studies exclusively involved volleyball players, whereas the third, Crossley et al., investigated athletes from varying sports such as volleyball, basketball, netball, soccer and tennis. As well as weight, Crossley et al. (2007) also demonstrated an association between body mass index (BMI) and patella tendinopathy. This was the case for both unilateral and bilateral patellar tendinopathy. This finding was similar to that of Malliaras et al. in 2003. Of the six studies included in the systematic review that looked at height, no study found an association between height and patellar tendinopathy (Lian et al., 1996; Witvrouw et al., 2001; Lian et al., 2003; Cook et al., 2004; Gaida et al., 2004; Malliaras et al., 2007). This suggests that the key variable may be weight. For this thesis, it was anticipated that the participants with patellar tendon disease would have a higher body mass than those participants with healthy patellar tendons.

In addition to weight, other anthropometric characteristics were studied. These include: BMI (because weight is one of the variables used to calculate BMI), waist-to-hip ratio, leg length difference, arch height of foot, quadriceps flexibility,

hamstring flexibility, quadriceps strength and vertical jump performance. However, the evidence for these risk factors being associated with PT was not strong and the authors argue that further studies are required before any conclusions could be made.

2.9.3 Playing position

Given that the relationship between position and PT in rugby players has not been studied, even in the recent Durcan et al. (2013) study, it follows that the risk factors of various rugby playing positions has not been studied either. Rugby athletes share skills that are common to athletes of other sports, which have a high prevalence of patellar tendinopathy. These include running, jumping and cutting. However, different positions within a rugby team would engage in different frequencies of these skills e.g. second row forwards being more likely to be involved in line-outs (Sankey et al., 2008); which often result in landing from six feet or more, and scrummaging, whereas the backs would traditionally be involved in more sprinting and cutting activities (Eaton & George, 2006). Forwards have also been shown to be generally taller and heavier than backs (Fuller et al., 2012).

A study by Lian et al. (2003) into patellar tendinopathy in elite male volleyball athletes found that there was a significant difference in the prevalence of PT according to the position played. They found that the prevalence of PT in outside hitters and middle blockers was significantly higher than utility blockers and setters. The authors argued that this could be explained by the higher number of maximal jumps performed by outside hitters and blockers than by other positions. As no data currently exists for rugby athletes, it is unclear whether the prevalence of patellar tendon disease differs according to playing position as it does in volleyball athletes. Therefore it seemed logical that another of the aims of this (Giles) study was to examine whether there is a relationship between player position and the presence of patellar tendon disease in rugby. It was anticipated that patellar tendon disease would be more prevalent in rugby forwards as these athletes are more likely to engage in maximal jumping activities (e.g. line outs) and are likely to have a greater body mass. The possible relationship between anthropometric risk factors and the prevalence of PT in rugby, for which there is little strong evidence, informed the

study design of this thesis.

2.9.4 Ground Reaction Forces

The first strategy in managing PT, that is identifying risk factors, has proven to be problematic. This is because the evidence is either weak, equivocal or absent. Turning now, to the second strategy, that of serial screening remains. As already discussed, the gold standard of screening, i.e. to ultrasound scan everyone, has drawbacks such as the expense and expertise required. What would be really helpful would be to identify a screening method that was simple to use, did not necessarily require qualified healthcare professional expertise and that could predict the presence of patellar tendon disease. Such a screening method may involve force plate analysis of landing activities. Force plates measure and record ground reaction forces (GRF). The action of a force (e.g. a rugby player's weight) on the ground receives an equal and opposite reaction force, this is the GRF (Porter, 2008).

It has been reported that athletes with patellar tendinopathy have altered lower limb landing strategies when compared to healthy controls (Richards et al., 2002; Bisseling et al., 2007). It is hypothesised that it is an increased patellar tendon load, and the inability to adapt to this increased patellar tendon strain, which predisposes to patellar tendinopathy.

Seegmiller & McCaw (2003) cite Sands et al. (1993) and report that the most prevalent type of lower limb injury in gymnasts is "repetitive stress syndrome injuries". To investigate whether the landing strategy of healthy gymnasts differed to that of healthy recreational athletes (and possibly predisposing to these injuries), Seegmiller and McCaw (2003) measured peak vertical ground reaction force (VGRF) and loading rate (LR) VGRF during a drop-landing protocol. The drop-landing protocol involved stepping off pre-determined heights of 30-, 60-, and 90-cm. The authors report that the gymnasts exhibited a 33% and 34% increase in VGRF, compared to non-gymnasts, when landing from a height of 60 cm and 90 cm respectively. There was no difference in the LR VGRF between the two groups of participants. The authors argue that the repetitive exposure to high loads during

landing may contribute to injury. Furthermore, Fietzer et al. (2012) described that dancers with patellar tendinopathy exhibited 36% greater peak VGRF during landing tasks when compared to their healthy counterparts. This notion concurred with Richards et al. (2002) who suggested that a high VGRF during landing tasks in indoor volleyball athletes might serve as a predictor of patellar tendinopathy.

In contrast, Bisseling et al. (2007) performed drop-jumps on 24 volleyball athletes from three different heights (30 cm, 50 cm and 70 cm). They found that there was no difference in the peak VGRF between athletes who had previous (but now asymptomatic) PT, recent patellar tendinopathy or healthy controls. In this study, Bisseling et al. used a clinical diagnosis of patellar tendinopathy and did not use imaging to confirm diagnosis. However, previous work by Cook et al. (2001) has suggested that a diagnosis of PT on clinical grounds alone is unreliable. Although there was no difference observed in peak VGRF in the Bisseling et al. (2007) study, the authors did observe a higher LR VGRF in the group who suffered previous jumper's knee ($p=0.05$) compared to controls. This led the authors to conclude that patients with previous jumper's knee used a stiffer landing strategy than healthy controls. This stiffer landing may be a risk factor in the development of patellar tendinopathy. These findings are similar to those reported by Edwards et al. (2010). Edwards and colleagues performed a stop-jump task on seven athletes with a PTA demonstrable on ultrasound (but no history or clinical signs of patellar tendon disease). These athletes were matched with seven healthy controls. The stop-jump task involved both a horizontal and a vertical landing phase. The authors found no significant between-group difference in the peak VGRF or the LR VGRF for the horizontal landing phase of the stop-jump task. However, during the vertical landing phase, the group with the PTA demonstrated a significantly lower LR VGRF compared with the control group ($P = 0.03$). There was no significance between groups for peak VGRF.

It is not clear whether differences in VGRF may pre-dispose to patellar tendon disease or be the product of adaptive changes in landing strategy as a result of the disease (Bisseling et al., 2007). Although the evidence remains equivocal in that one study has been identified which found peak VGRF would not be useful in identifying athletes suffering from, or at risk of patellar tendon disease, there is more evidence

that it may be useful in this regard. If VGRF from landing tasks were found to be a valid method of screening for patellar tendon disease in rugby, this efficient and easy-to-use test could identify those with patellar tendon disease, or those at risk. This would enable appropriate management of the condition so preventing the requirement to take prolonged time off from competition or even early retirement. Therefore, the final aim of the study was to begin to investigate whether players with patellar tendon disease (symptomatic or asymptomatic) exhibited different VGRF and LR VGRF than controls and whether logistic regression could predict tendon status in rugby athletes i.e. whether there is patellar tendon disease or not (regardless of symptoms). Were this possible, identifying players with asymptomatic patellar tendon disease would allow suitable preventative measures to be put in place before symptoms arise, avoiding injury progression and the need for time off from training and competing.

2.9.5 Previous injuries

To date, there appears to be no studies suggesting that previous injury to the knee may be a risk factor for development of PT. Empirical studies of risk factors for PT place previous knee injury as an exclusion criterion, because of its potential to be a confounding variable.

2.9.6 Genetics

Although it is accepted that the aetiology of tendinopathy is likely multifactorial, recent studies have suggested that genetics may play a role in predisposing individuals to the development of tendinopathy. Several studies have investigated the association between the ABO blood groups and tendinopathy. However, the association between blood group O and rupture of the Achilles tendon ($P = 0.03$) is stronger than for blood group O and chronic tendinopathy ($P = 0.1$; Kujala et al., 1992). These results support those found in 1989 by Jozsa et al. in a population 832 patients with tendon rupture. Conversely, these results have not been replicated in more recent studies (Leppilahti, Puranen & Orava, 1996; Maffulli et al., 2000).

A review in 2007 (September, Schwellnus & Collins) summarizes the current evidence for a genetic component to the development of tendinopathy. This evidence mainly involves two genes, Tenascin-C (TNC; Mokone et al., 2005) and COL5A1 (Mokone et al., 2006). These original studies involved Caucasian athletes (individuals with Achilles tendon injuries and a control cohort) from a South African population. The findings have since been replicated in an Australian Caucasian population with similar results (September et al., 2009). September, Schwellnus & Collins (2007) propose that screening for variants in these genes (TNC and COL5A1) may have a role in identifying those individuals at risk of developing tendinopathy. However, the authors acknowledge that more research is needed before such a screening service can be offered.

2.10 Conclusions

In conclusion, PT is a common condition among sporting populations and is thought to be mainly degenerative in nature. According to Cook and Purdam (2009), there are three stages: reactive tendinopathy, tendon dysrepair and degenerative tendinopathy. Whilst recovery from reactive tendinopathy is possible with appropriate management, this becomes more difficult with tendon dysrepair and limited when the stage of degenerative tendinopathy is reached. The consequences for an athlete who is not appropriately managed can be career limiting or even career ending. The development of the condition can be insidious and difficult to link to any particular match or training episode. Furthermore, patellar tendon disease may be present in the absence of symptoms. However, it has been demonstrated that athletes who have asymptomatic patellar tendon disease are at increased risk of developing future disabling symptoms of patellar tendinopathy.

There are several studies of PT in sports that involve similar skills to rugby. Whereas prevalence rates vary, there is sufficient evidence that PT is a recognised consequence of playing these sports. Very few studies investigating the prevalence of PT in rugby exist which was one of the reasons for this (Giles) study. Those studies that do exist are problematic for several reasons. The definition of PT used in studies

has led to under-reporting of the condition. There is a lack of consistency as to whether incidence or prevalence is reported (incidence is not recommended for chronic overuse injuries) and problems with coding have resulted in ambiguity. A recent study (2013) by Durcan et al. (reported after this thesis began) demonstrated a high prevalence of patellar tendon disease in academy rugby players. Other prevalence studies in rugby have significant flaws in the methodology so the results cannot be relied upon.

Patellar tendinopathy is a condition that is frequently written about. However, when undertaking a systematic review, Van der Worp et al. (2011) found only eleven studies which met their inclusion criteria. None of these studies included rugby athletes. The conclusion of the systematic review was that there was not a strong relationship between various anthropometric characteristics and PT, apart from weight for which there was a reasonably strong association. In team sports other than rugby, an association between PT and playing position has been demonstrated. The methodology for this (Giles) study was designed to begin to address the lack of data on these relationships for rugby. It was hoped that the risks to rugby players of developing patellar tendinopathy could be better understood.

There is an accepted gold standard for diagnosing patellar tendon disease. This includes clinical examination accompanied by ultrasound imaging of the patellar tendon. The difficulties associated with providing both tests on a comprehensive and regular basis have been described. There is some evidence from other sports that altered VGRF measurements from landing tasks would be a proxy measure for the presence of PT. Drop-landing tasks are easy to perform and if the VGRF measurement was predictive of patellar tendon disease in rugby, it would allow comprehensive and regular screening of rugby athletes. In turn, this could lead to appropriate management, prevention of further degeneration and preservation of the athlete's career. The study design therefore included an investigation of whether force plate analysis of drop-landing tasks could predict the presence of patellar tendon disease.

Chapter 3: Methodology

3.1 Introduction

The study design is informed by the conclusions of the literature review (chapter 2), the researcher's personal experience as a medical officer to a rugby club, and personal communication with other sports doctor colleagues about the prevalence of patellar tendon disease in rugby: the relationship between patellar tendinopathy and various risk factors, and the extent to which the presence of patellar tendon disease can be predicted using analysis of ground reaction forces. In order to address these issues, the study design has a number of aims and objectives, which are set out below.

3.2 Aims and Objectives of the study

Prevalence

To estimate the prevalence of patellar tendon disease in rugby players, the following objectives were set:

1. To determine the prevalence of symptomatic patellar tendinopathy (PT) in a sample of rugby union athletes
2. To determine the prevalence of asymptomatic patella tendon disease seen on ultrasound imaging in a sample of rugby union athletes.

Patellar tendon disease and risk factors

In order to understand the relationship, if any exists, between patellar tendon disease and anthropometric factors and playing position, the following objectives were set:

3. To investigate whether rugby athletes with patellar tendon disease have a greater mass than rugby athletes with healthy tendons.
4. To investigate whether playing position affects the prevalence of patellar tendon disease

Prediction of patellar tendon disease

In order to investigate whether it is possible to predict the presence of patellar tendon disease using force plates, therefore avoiding the need for ultrasound screening of high numbers of athletes, the following objectives were set:

5. To investigate whether rugby athletes with patellar tendon disease have different vertical ground reaction forces when performing drop-landing tasks than controls
6. To investigate whether force plates measuring vertical ground reaction forces from drop-landing tasks can predict tendon status during a landing task i.e. healthy or patellar tendon disease

3.3 Study Design

To deliver the study's aims and objectives, a multi-method approach was considered necessary. The design of the study entailed the following:

1. Recruitment of a sample of rugby players
2. Collection of data on each study participant's general characteristics
3. Collection of baseline symptom score on each study participant's
4. Examination of study participants, clinically and with ultrasound, for signs of patellar tendon disease
5. Performing a drop-landing task to measure vertical ground reaction force (VGRF) and Loading rate VGRF (LR VGRF)
6. Undertaking statistical analysis to:
    a. Estimate the prevalence of patellar tendon disease
    b. Examine the relationship, if any exists, between patellar tendon status and weight and playing position
    c. Examine the relationship, if any exists, between patellar tendon status and VGRF and LR VGRF
7. In order to be able to undertake data analysis, it was necessary to categorize participants into one of the following groups:

a. Asymptomatic; no ultrasound changes (the control group)
b. Asymptomatic; positive ultrasound changes (comparison group 1, hereafter named "PTA group")
c. Symptomatic (comparison group 2, hereafter named "PT group")

Each of these study design features is discussed in detail below.

3.4 Ethical Approval

As the research involved human subjects, ethical approval was sought from the University Research Ethics Committee. The main ethical considerations were:

That participants would be assured of anonymity and confidentiality. That is, that none of their personal data or individual findings would be disclosed to a third party without the participant's expressed consent. Each participant was given a unique identifier (known only to the researcher), which was stored separately from the participant's name. This meant that it was not possible to identify any player by the results.

That all data were stored on a password protected file. Only the research team knew the password to this file. This allowed for adequate protection of information whilst also enabling verification of findings (if needed).

That each participant would give informed consent to be involved in the study. This means that the study's aims and objectives, and data collection and handling processes would be explained to the participant in terms that they could understand. It also meant that participants would be given an opportunity to ask questions for clarification on any point. As part of informed consent also, each participant would be informed that they could withdraw from the study at any time and request that any of their personal data be destroyed. In addition, they would be informed that no third

party would be informed of their withdrawal.

That any discovery of PT previously undiagnosed would be handled appropriately. This meant that at an appropriate time participants would be informed of the findings and advised of the diagnosis and that this should be shared with the coaching / health professional team so that appropriate management could be instigated. However, it was made clear that the decision to do this would rest with the participant.

An information leaflet (appendix 3) was devised which set out all of the above details and was provided to each participant. Before any data were collected, an informed consent form (appendix 4) was signed. These issues were addressed in this way in order to comply with relevant legislation e.g. Data protection act 2008 and sound ethical principles on conducting human research as set out in the current Helsinki declaration of human rights (World Medical Association 2008).

3.5 Sample

If the hypothesis that there is a high prevalence of patellar tendon disease in rugby players was correct, then it was anticipated that the data would fall in to two groups. The first group would have healthy patellar tendons and the second group would have patellar tendon disease (this second group could be further subdivided into those who had clinical signs of the disease, and those who were clinically silent). The differences between the two main groups, in terms of a number of variables (mass, playing position etc.), would then be analysed. In doing this, it would be important to analyse the size of the difference between the two groups. For this study, this would include understanding the level at which the difference in mass affected tendon status and what effect size in VGRF would be indicative of the presence of tendon disease. When this study commenced, there were no published studies in rugby which have examined the relationship between these variables and patellar tendon disease, it meant that there was not any data regarding distribution, standard deviation etc. of these variables in rugby players with patellar tendon disease. The absence of these data would make it very difficult, if not impossible, to state likely effect size in

VGRF due to disease. For this reason, it was felt that it was not appropriate to undertake power analysis to determine the sample size needed to examine the differences between the two groups.

It was, however, hoped that this study would provide sufficient data for future studies about prevalence (as noted above), and the distribution (including standard deviations) of all the key variables, and in particular what difference in effect size could be anticipated between a group of healthy players and those with patellar tendon disease. In turn, this would enable power calculations to be undertaken for future studies. It might be that any differences that would be material would be subtle, and if this were to be the case, a large sample size would be required. However, at the start of this study, there was no robust data about prevalence of patellar tendon disease in rugby athletes. It would be unethical to subject a large number of players to testing (even though testing would be non-invasive) when the presence or absence of the key variable was not known, i.e. was patellar tendon disease prevalent or not?

It was decided that age-grade players (those still eligible to play for an under-20 team) would be targeted for inclusion in the study: Access was more straightforward as the season was already underway during the data collection period and it was considered that age-grade rugby players would have less playing, training and sponsorship commitments than senior players. As is discussed fully in chapter 5, it transpired that age-grade players might have greater training commitments than the senior squad, which causes problems when extrapolating results from the sample to the senior squad. In addition, at this stage there was no certainty of the prevalence of patellar tendon disease or that a relationship between VGRF and PT existed (a proof of concept had yet to be established). Depending on the findings, the study could be extended at a later date to include senior squads.

3.6 Inclusion / Exclusion Criteria

1. All participants had to be free from any acute back or lower limb injury (not including patellar tendinopathy) i.e. each study participant had to be undertaking a full competitive training regime as advocated by Bisseling et al. (2007) who report that any acute injury may affect biomechanical properties of movement tasks and have a confounding effect on results. In addition, the inclusion of players with back or lower limb injury might expose the player to unnecessary risk (this is discussed in more detail in ethical considerations). At any one time, approximately 25% of rugby squads are injured (Holley, 2010), which reduces the total possible sample size.

2. All study participants had to have been playing competitive rugby for at least 5 years prior to enrolment in to the study. This was to ensure that study participants were established rugby athletes and had experienced the typical physical stresses of rugby matches and training practices. The literature is silent on the length of playing time required for sample selection that would represent a typical rugby population (it transpired that none of the athletes included in the study had played elite rugby for less than 5 years).

3. Only those players aged 18 years and over at the time of recruitment were offered the opportunity to participate, i.e. a player of exceptional ability who at 17 years of age may be representing an under 20 squad was not invited to participate. It would have compromised the study to include players under the age of 18 (even if they were representing an under 20 squad and had played competitive rugby for five years) because of the risk that the player may have had Osgood–Schlatter and/or Sinding-Larsen-Johansson syndrome which occurs in the adolescent athlete. Both these conditions result in symptoms that affect the extensor mechanism of the knee (Brukner et al., 2007) and can mimic patellar tendinopathy. Exclusion of participants based on the possibility of these conditions being present is also advocated by Malliaras et al. (2007). Informed consent and other safeguarding procedures were more straightforward for individuals over the age of 18 years of age.

The total population of under-20 elite players in Wales is circa 80. This compares with Ireland, another UK country with the same number of regions (four) as Wales (Durcan et al., 2013). It was decided, therefore, that the drawn sample would be all regional academy players in Wales under-20 years of age (but over the age of 18 years). Given that the estimation that 25% of players in a squad are injured at any one time, this probably means that the potential total sample size is circa 60 players. Nevertheless, because 25% is an estimate, 80 was taken as the total possible sample size for analyzing response rates.

3.7 Recruitment Strategy

Following approval for the study from University's Human Research Ethics committee, contact was made with each of the four regional rugby clubs in Wales inviting their age appropriate under-20 academy athletes to participate. Initial contact was made via an information letter (or email) distributed to coaching and/or medical staff at the respective rugby club (appendix 2). The letter contained the study's details and a statement seeking permission to invite their athletes to take part. Once permission from the club representative was granted, visits were made to each region to discuss the opportunity with the coaches and the potential players involved. Recruitment letters and information leaflets (appendix 3), which were approved by the University Ethics Committee, were offered to potential participants. Where visits could not be arranged in a timely manner, dialogue took place via telephone conversation and/or email. Formal invitations to participate in the study were made to the players directly. As initial response was poor, it was decided that we would request the club physiotherapists to invite the participants into the study. Contact information was offered so that any player (and/or coach) could seek further information.

Before participation within any part of the study commenced, each participant had completed and returned written informed consent using consent forms approved by the University Ethics Committee (appendix 4).

3.8 Baseline demographic data

The demographic data collected were limited to that which was necessary to meet the study objectives. Numbers of players per region are available (see results section). However, cross-tabulation by region was not undertaken for several reasons:

1. The sample size per region was too small to provide meaningful results
2. The results would not have informed the study objectives
3. An undertaking of confidentiality was given to players and the regions. Presenting data by region would breach confidentiality and might have commercial consequences for the region. Furthermore, breach of confidentiality could result in resistance to participation in future research. However, individual reasons were given their results.

Data on the player's age, stature, weight, favoured playing position and dominant foot for kicking were collected. Stature was measured with the athlete barefoot using a standard stadiometer and recorded to the nearest centimeter. Body mass was measured using a commercially available force plate (400 Hz: Accupower Plate; Amti Advanced Mechanical Technology Inc., MA, USA) connected to a computer. During measurement, the force plate was zeroed, and the participant instructed to step on to the force plate, to stand upright in the centre of the force plate and to face forward. The weight of the participant was recorded in kilograms (rounded to one decimal place e.g. 96.7kg).

Study participants were asked their favoured playing position and their dominant foot. Favoured playing position was defined as the position on the rugby field where they played the majority of their competitive rugby games. Dominant foot was defined as the foot they would use if they wanted to kick a rugby ball the greatest distance. Study participants were asked to reveal whether they had ever had a previous diagnosis of patellar tendinopathy or "jumper's knee". This question was asked at the beginning of the data collection procedure in order to avoid the risk of interviewer bias.

3.9 Baseline symptom score (for use as a clinical management tool if PT or PTA was diagnosed)

One of the key ethical considerations (which are discussed in detail below) was what to do if PT was diagnosed during the study. It would be unethical not to offer the player referral for appropriate treatment and further investigations by the health professionals in the player's region. The only questionnaire widely used to assess the symptoms of diagnosed patellar tendinopathy is the VISA-P score. The VISA-P score (appendix 5) would provide the baseline from which the player's own regional health professionals could monitor the effect of subsequent treatment. It was devised by the Victorian Institute of Sport in Melbourne with the primary aim of assessing the severity of symptoms and functional status of those patients with patellar tendinopathy (Visentini et al., 1998). It is a self-administered questionnaire that is widely used to monitor response to treatment in established patellar tendinopathy. It has been shown to be a reliable and a valid tool for determining whether athletes experience symptoms from their already diagnosed patellar tendinopathy (Khan et al., 1999; Cook et al., 2000). This was important so that we could be confident in the content of any referral made. The VISA-P has benefits over other scoring systems (such as the Cincinnati Sports Activity Score or Visual Analogue Scales) as it was specifically designed to assess functional status for those with patellar tendinopathy and not lower limb injuries in general.

VISA-P is not intended as a research tool but as a clinical tool to assess progress of management following diagnosis. The VISA-P questionnaire is one intended for players to complete. That is self-report. There is always a risk in any self-reporting tool that respondents will "fake good" (Streiner and Norman, 2000) or provide what is perceived to be "socially desired responses". For example, the cause of faking good might be to avoid missing training or playing (and potentially incurring financial loss). In the case of socially desired response, the player may not wish to let down either his team mates or coaching staff: or perceive the symptoms to be a sign of weakness within a highly competitive environment. This means that there is a risk of obtaining false negatives if the VISA-P was used as a screening tool for patellar tendon disease. For these reasons there is a risk of using a self-reporting tool of this kind for research purposes. A reliable and valid research tool is designed in such a

way that such potential biases could not influence the responses given.

The VISA-P scores form part of the ethical integrity of the study i.e. they could be used to provide a symptom baseline for further management. It was initially decided not to use the VISA-P questionnaire as a screening tool. The total possible VISA-P score is 100. Points are deducted according to symptoms identified. To be used as a screening tool, it would be essential to know at what VISA-P score a player should be referred for clinical examination and ultrasound screening of their patellar tendons. This would entail very detailed scaling work to attempt to identify a threshold for referral and, in any event, such an outcome may never prove possible because VISA-P is designed as a tool for use with those who already have symptoms. If it were possible to identify a threshold, the issue of faking good would undermine the tools usefulness. Having said that, this hypothesis was tested by undertaking a cross tabulation of VISA-P scores with the presence or absence of disease.

It was decided that the researcher would assist the players in completing the VISA-P questionnaire. This would ensure a higher response rate than would be the case if the questionnaire were given with the request that it be returned by post. This approach would also ameliorate the difficulties associated with any potential poor literacy levels. Again the rationale for taking such care over this issue was to ensure that any clinical condition did not deteriorate because it had not been drawn to the attention of both the player and their health professionals.

3.10 Clinical Examination

Each player (n=46) received an assessment by an experienced sports medicine physician (appendix 6) who had no knowledge of their previous medical history or the symptoms that may have been recorded on the players VISA-P score. The clinician asked simple questions about pain and tenderness experienced during examination. The clinician did not ask questions concerning pain and tenderness experienced during training and competition. This meant that players were categorised according to clinical findings only.

The examination for each player followed a standard protocol with specific focus on looking for signs of patellar tendinopathy i.e. pain to palpation over the inferior pole of the patella and pain at the insertion of the patellar tendon when performing functional exercises. This ensured an element of consistency. Following the approach advocated by Brukner et al. (2007), the clinical examination started with the participant standing with legs adequately exposed. The participants were asked to undergo functional exercises, which involved loading the extensor mechanism of the knee to try to reproduce the pain associated with patellar tendinopathy. These included hopping on one foot, lunges, squatting and single leg squatting (figure 3.1). A total of 10 repetitions for each exercise were performed i.e. ten repetitions for each leg when performing a single leg exercise.

Figure 3.1: Study participant undergoing lunges

Examination continued with the participant lying supine on an examination couch. In this position the clinician palpated for tenderness over the inferior pole of the patella and at the insertion of the patellar tendon. This was performed both in full extension and with the knee in slight flexion (approximately 20 degrees; figure 3.2). Palpation

of the patellar tendon is highly sensitive at reproducing the pain of patellar tendinopathy (Cook et al., 2001).

Figure 3.2: Examiner testing for tenderness over the insertion of the patellar tendon

It is essential that the data collected on clinical examination was consistent. As there was only one examining clinician, there was no need for inter-rater reliability analysis. A pro-forma was used by the examining clinician to record whether there was tenderness on palpation of the patellar tendon or it's attachments and whether the athlete experienced pain on functional testing. Some researchers (Cook et al., 2001) have assessed pain by severity using points on an analogue scale. It was decided that such an approach was not relevant for this (Giles) study because a. pain symptoms had been recorded using the VISA-P score, b. the sample size was too small to have safe calculations of cross-tabulations of grades of pain, c. each athlete would receive an ultrasound scan as part of the data collection procedure.

The pro-forma used by the examining clinician in this study can be found in appendix 7. The final item on the pro-forma required the examining clinician to record the presence or absence of clinical findings i.e. a binary choice. This forced the clinician to make a firm commitment about their clinical impression and it

avoided the need for post-hoc coding of clinical narrative. The format also facilitated data entry.

3.11 Ultrasound imaging

In the hands of an experienced clinician, ultrasound allows visualisation of the patellar tendon and this together with the clinical examination determines with a very high degree of accuracy whether tendinopathy is present. As ultrasound is currently the gold standard in diagnostic imaging of patellar tendinopathy (Warden et al., 2007), no testing of validity and reliability was needed on this data collection tool. It also has the benefit over other types of diagnostic imaging (e.g. CT scanning) because it does not expose the participant to any ionising radiation.

All participants (n=46) received an ultrasound scan of their patellar tendons. Both left and right-sided tendons were scanned on each participant. All ultrasound scans were performed by the same examiner, who was experienced in the field of musculoskeletal ultrasound and uses the technique regularly in orthopaedic clinics and in his role as a sports physician to a Welsh rugby region (appendix 6). Again, using one clinician to perform the ultrasound scans has the advantage of avoiding the need for inter-rater reliability testing. The drop-landing task was performed after the ultrasound examination. This approach was to ensure an element of blinding to the examiner performing the ultrasound scans, i.e. the examiner would not know the result of the drop-landing tasks prior to performing the ultrasound scan and could not be influenced by the results.

Ultrasound data were recorded using high-resolution grey-scale ultrasound (256 grey shades) with the aid of colour power doppler (256 colours) and a 7 MHz linear array transducer (Sonosite MicroMaxx, Washington, USA). For image optimization, the focal depth was adjusted to correspond with the level of the patellar tendon. For colour power doppler, flow sensitivity was set to “medium”. The scans were viewed on the 21.34 cm x 16 cm display. Contrast and brightness settings on the display were adjusted corresponding to room conditions to allow clear visualisation of the image.

In preparation for the ultrasound scan, each player lay at an angle of 45 degrees on an examination couch with both limbs adequately exposed (see figure 3.3). This allowed an adequate position for imaging of the tendon and also the optimum comfort of the participant. During examination, a pillow was placed under the knee of the tendon being scanned. This placed the tendon under a slight degree of tension and allowed for clearer visualisation (as advocated in Warden et al., 2007). Water-soluble gel (Aqua-gel) was applied to both the transducer and over the region of the patellar tendon. This was to aid diagnostic coupling by removing direct contact with the skin and again allowing for better visualisation of the tendon (Sonosite, 2005).

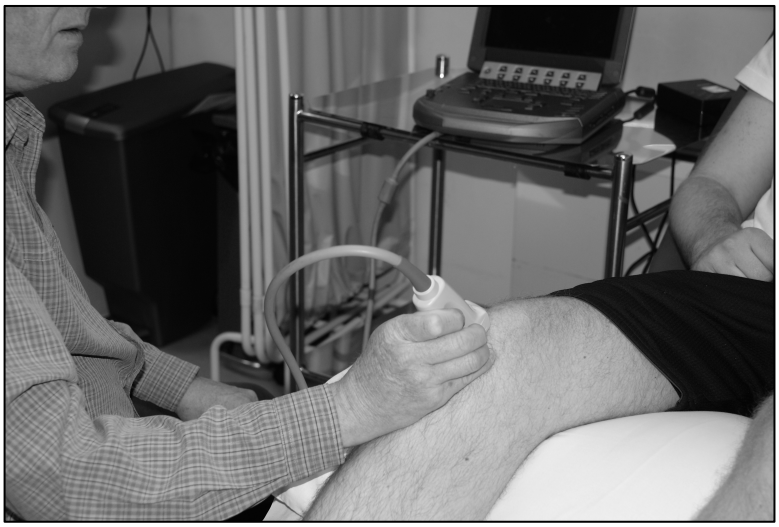


Figure 3.3: Position of the participant for ultrasound scanning of the patellar tendon

Transverse and longitudinal scans of the patellar tendon were performed. Figures 3.4 and 3.5 demonstrate the orientation of the transducer used to capture the longitudinal and transverse imaging respectively.

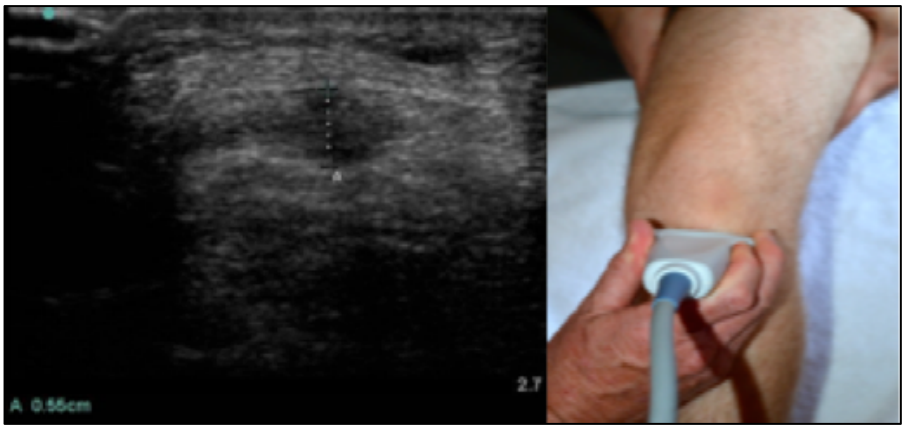


Figure 3.4: Orientation of the ultrasound transducer to visualise the tendon belly in the transverse plane

Figure 3.5: Orientation of the ultrasound transducer to visualise the tendon in the longitudinal plane

The aim of the ultrasound examination was to identify a PTA. A PTA was defined as the presence of a hypoechoic area seen in both the longitudinal and transverse scans. An example of a PTA can be seen in Figures 3.4 and 3.5. This definition is consistent with that used in other studies (Cook et al., 2000; Cook et al., 2004; Gisslèn et al., 2007 and Warden et al., 2007). The question of grading the ultrasound changes was considered. Cook et al. (2000) concluded that the size of a PTA had not yet been shown to correlate with disease or symptom severity. In 2013, in a study of both Achilles and patellar tendons, Comin et al. reported that moderate or severe hypoechoic areas are weakly predictive only for future symptoms of tendinopathy. Moderate or severe were not defined in their paper. This would suggest that at the

moment, presence or absence of a PTA should be the only criterion used to classify the presence of disease. Even though, Archambault et al. (1998) advocated a grading system containing three points (1=normal, 2=tendon thickening, 3=hypoechoic area, regardless of size), Comin et al. (2013) found no correlation between any sonographic abnormalities (including tendon thickening) and the development of symptoms, apart from the presence of hypoechoic areas. For this thesis, it was decided that tendons would be considered abnormal (show signs of tendinopathy) if a PTA was present (regardless of size). The rationale for this is that any further granularity has not been shown to be of material value in either diagnosis, disease trajectory or symptom control.

It was decided that the options available to the examiner were binary, i.e. whether there was a hypoechoic area or not. This ensured that data collection was as objective as possible and that the examiner made a commitment to their diagnosis. This also facilitated coding. All tendons in this study considered to have a PTA, had clear hypoechoic areas illustrated in both the longitudinal and transverse scans. The pro-forma for data collection can be seen in appendix 8. It was decided that presence of neurovascularisation would be recorded (and could be communicated to the player's physiotherapist) but would not be used to determine group status in the study. This is because the majority of tendon studies simply use the presence or absence of a PTA to determine the health of the tendon (Cook et al., 2000; Cook et al., 2004; Gisslèn et al., 2007).

Once an uninterrupted view of the patellar tendon was visualized, the image was saved to the hard disk of the ultrasound machine using the participants unique identifier followed by the letters L or R to distinguish which patella tendon i.e. left or right. The process was then repeated for the contralateral leg. Following data gathering, the images were transferred to a password-protected file on a University user account and saved in JPEG format.

3.12 Drop-Landing Task

All participants (n=46) undertook a drop-landing task and force plate analysis to assess their peak vertical ground reaction force (VGRF) during a drop-landing movement. Ground reaction force data were collected using a commercially available force plate (400 Hz: Accupower Plate; Amti Advanced Mechanical Technology Inc., MA, USA) connected to a computer. Flight time (FT), contraction time (CT) and FT:CT ratio were captured using software (Accupower Software; Amti Advanced Mechanical Technology Inc., MA, USA), and exported to Excel (Microsoft) for analysis. Portable force plates have been shown to be both valid and reliable in measuring force-time data in jump and landing tasks (Walsh et al., 2006). Portable force plates were chosen because they are more accessible to training athletes and sporting teams.

Study participants wore socks and training shoes for the data collection procedure. This minimized the risk of injury and had the added advantage of enabling comparison with other studies of sports in which jumping is a key feature e.g. basketball and volleyball players (as discussed in chapter two).

The procedure involved performing a drop-landing from a 30 cm high platform situated immediately behind the portable force plate. The height of 30 cm was relative to the force plate (see figure 3.6). A drop-landing was used as opposed to a jump so that the test height for each player was standardized. If a jump protocol had been used, adequate comparisons between different players would not be able to be made. This height would also allow adequate comparison to be made with data from other studies (e.g. Seegmiller & McCaw, 2003; Bisseling et al., 2007). Although Bisseling et al. (2007), in addition to a landing from a height of 30 cm, also used a landing from a height of 50 cm and 70 cm in a sample of volleyball players. It was decided that at this exploratory stage of investigation, where the risks of performing landing tasks in rugby players with patellar tendinopathy were not known (and given that the prevalence of patellar tendinopathy was not known), the height that was thought to pose the least risk to players was chosen i.e. 30 cm.

Figure 3.6: Demonstrating set up of equipment for drop-landing task

Players were instructed to step off the test platform with their test leg (left or right) first. They were instructed to land facing forward, with one foot on the force plate (test foot) and the other (non-test) foot parallel to the test foot. The participants were encouraged to land as naturally as possible (without rebounding or stumbling). After landing, the participant was advised to hold their stance for 10 seconds. This was to ensure that the anticipation of walking away from the test area did not cause the player to stumble. A random number generator was used to decide which leg would be the test leg for the first landing task. The drop-landings were then completed in block order, that is, if the random number generator indicated that the left leg would be the test leg first, then all four jumps for the left leg would be recorded first. This approach was adopted as to avoid the need to re-arrange the testing equipment between consecutive jumps. There was a rest period of 60 seconds between consecutive jumps, measured by an electronic countdown. This was to eliminate the influence of fatigue on results. It has been suggested that fatigue may increase patellar tendon loading by an inability of the lower limb to dissipate efficiently the loads sustained during landing tasks (Steele et al., n.d.).

Each drop-landing was witnessed by the same observer. Before data collection, each participant was allowed to practice the landing protocol until both the participant and the observer were confident with the procedure. The observer recorded the landing

technique making a note that: one foot landed on the force plate, there was no significant stumble after landing. If the observer believed that there was any fault with the technique, the participant was asked to repeat the landing and the data from the "faulty" landing discarded. This happened once during the study.

Peak Vertical Ground Reaction Force (VGRF [N]) and time to peak VGRF (tVGRF [ms]), data were obtained. The tVGRF was defined as the temporal difference between VGRF and Initial foot-ground contact (IC). IC was defined as the time when the VGRF first exceeded 50 N. The Loading Rate vertical ground reaction force (LR VGRF) was defined as the peak VGRF value divided by the tVGRF. The graph below is illustrative of the data produced by the computer software following a drop-landing (figure 3.7).

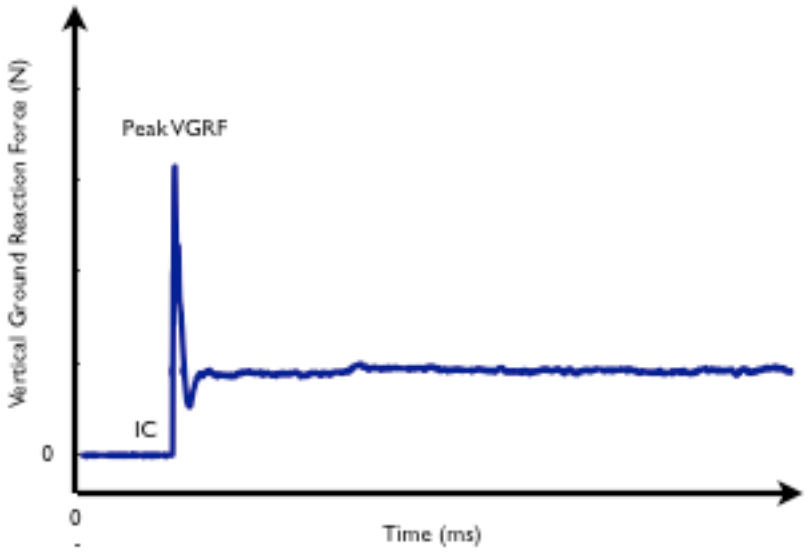


Figure 3.7: Graphical representation of acquired ground reaction force-time curve for drop landing task. IC, initial foot-ground contact; Peak VGRF, peak vertical ground reaction force

Each participant performed the drop-landing four times in total for each leg, the first landing for each leg being treated as a practice run and the data being disregarded.

The profile for each landing task was saved as an electronic copy to the computers hard disk. Each profile was saved using the participants unique identifier, followed by a letter representing the leg being measured, followed by the drop jump attempt i.e. 040186L(1) or 040186L(2) for a study participants first or second jump respectively. The letter L indicates the left leg was measured. This allowed for adequate protection of information and could be used for verification of findings (if needed).

3.13 Statistical Analysis

Data were analysed using IBM SPSS Statistics for Windows (version 21.0.Armonk, NY: IBM Corp.). The purpose of the statistical analysis that was carried out was geared toward answering the study questions. Participants were split into three groups. The first group contained players who had no clinical signs of PT on examination, and had tendons that were ultrasonographically normal (control group). The second group contained players who had no clinical signs of PT on examination. Their ultrasound scan demonstrated a PTA (PTA group). The third group contained players who had clinical examination signs consistent with PT. The ultrasound scan of players in this group also demonstrated a PTA (PT group).

Overall prevalence of PT and PTA was determined using descriptive statistics with mean and standard deviation reported. The weight in the three experimental groups was compared using the Kruskal-Wallis test. This test was used rather than a parametric test because the study sample is small (see chapter 4) and the distribution of weight across the sample appeared to follow a bimodal distribution and not a normal distribution (presumably due to the two distinct groups in the sample: forwards and backs). In this instance, a non-parametric test is the more appropriate test to be used. The non-parametric test used to describe the difference between three or more conditions is the Kruskal-Wallis test (Dancey et al., 2012).

The prevalence of PT and PTA was compared between the forwards and backs using chi-squared tests (χ^2). This test was used because playing position is a categorical

variable, that is, participants could be classified as a forward or a back but could not be both. The chi-squared test is appropriate when assessing association between categorical variables (Dancey et al., 2012).

The difference in peak VGRF and LR VGRF in the three experimental groups (control, PTA and PT) were compared using Kruskal-Wallis test. For this analysis, the subjects were tendons rather than players. It was decided to treat each tendon as a variable. Logistic regression was used to investigate whether ground reaction force data could be used to predict tendon status i.e. healthy or patellar tendon disease (PTA and PT groups combined). PTA and PT were grouped together for this analysis for two reasons. The first being that the sample size is relatively small. The second being that, if VGRF could be used to predict tendon status, it would not matter whether the athlete was symptomatic or not. This is because asymptomatic PTA is at risk of progressing to symptomatic PT (as already discussed).

Chapter 4: Results

4.1 Introduction

In this chapter, the results that are presented relate to the main aims and objectives of the study. A summary of any statistical analysis is included. A discussion of the results in relation to the appropriate literature can be found in chapter 5.

4.2 Sample

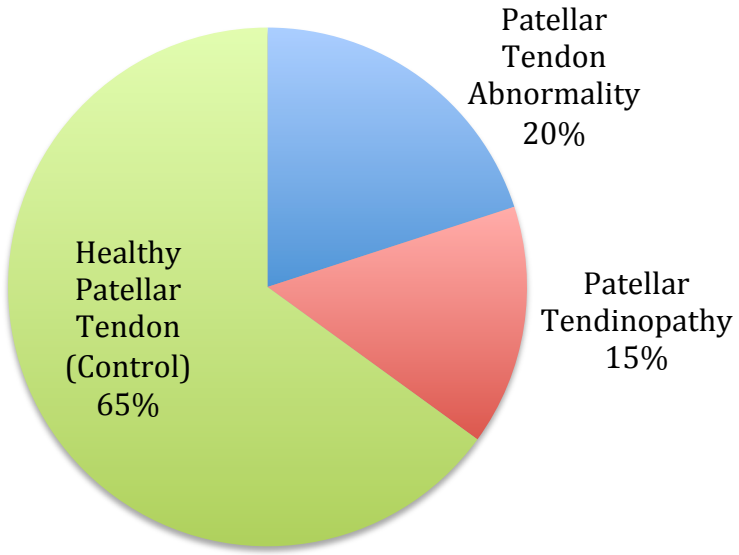
There are approximately 80 age-grade academy players in Wales. Only those who met the inclusion criteria i.e. injury free, were invited to participate. Team physiotherapists provided the names of those players who were both available and injury free. Of those, 46 players participated in the study (Blues n=16, Ospreys n=15, Scarlets n=11, Newport Gwent Dragons n= 4); this gives a minimum response rate of 57.5%, as physiotherapists did not provide data regarding the number of ineligible players. In order to increase the sample size, arrangements were made to include a further 23 players from an English region. Unfortunately on the day scheduled to test both the English region (and a follow up visit to the Newport Gwent Dragons academy) the force plate was damaged and it was not possible to reschedule. This has resulted in a smaller sample than desired, which is frustrating because a larger sample may well have resulted in statistically significant results.

Of the 46 participants, 21 were forwards and 25 were backs. All participants were right footed. There was an average stature of 1.83 m (standard deviation 0.06), body mass of 94.3 kg (11.475) and BMI of 27.9 (2.7). The average VISA-P score was 92.9 (14.5).

4.3 Prevalence

A total of six players (13%) reported that they had been previously diagnosed with patellar tendinopathy or "jumper's knee". Of these six, four were in the PT group, one in the PTA group and one in the Control group. The distribution of results according to clinical status is exhibited by chart 4.1.

Chart 4.1: Distribution of results according to disease group



No player had bilateral symptomatic patellar tendinopathy. In total, 21 tendons had a PTA (12 left, 9 right). All patellar tendon abnormalities in this study were in the proximal pole of the tendon.

VISA-P

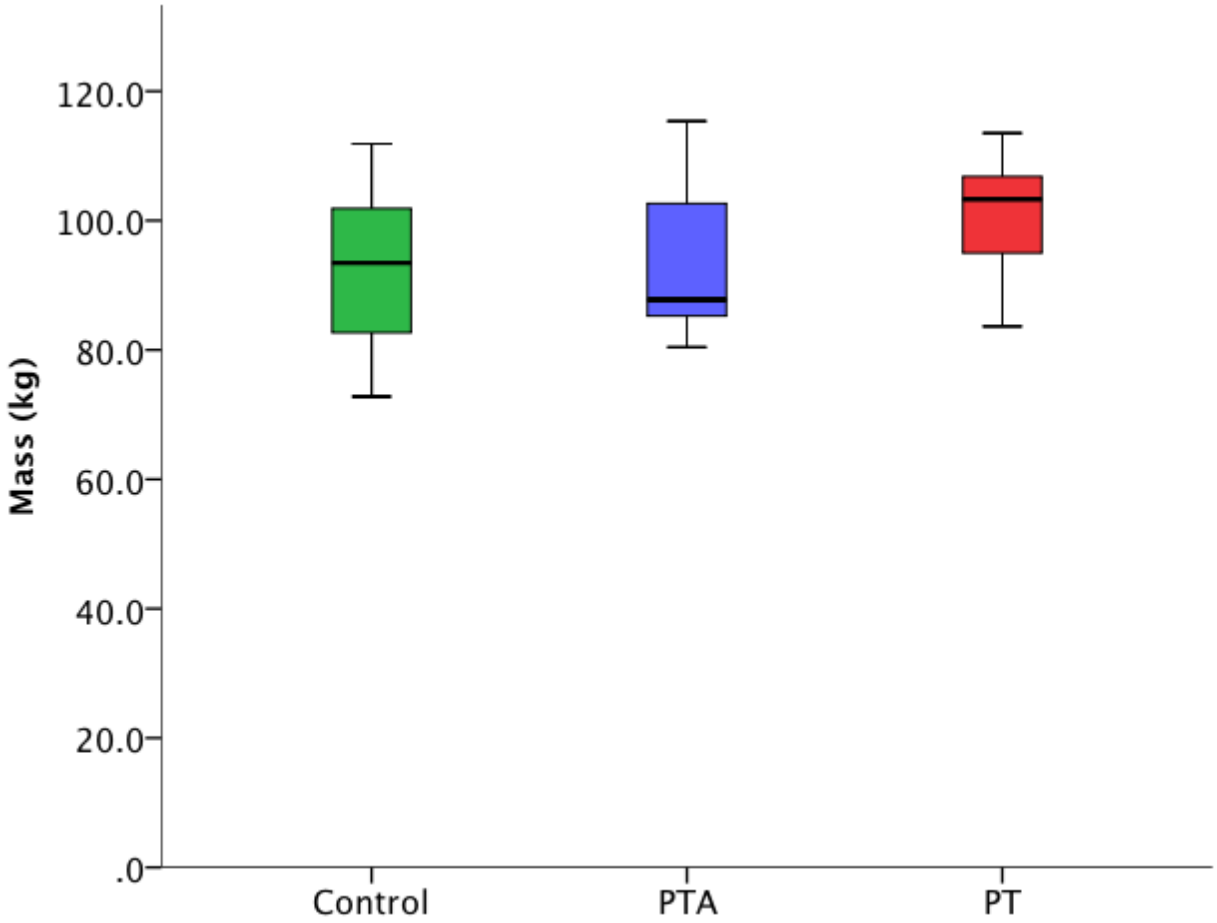
The average VISA-P score differed according to group status: control 99.0 (3.1), PTA 91.2 (11.4), PT 68.9 (22.4). A Kruskal-Wallis test showed that there was a significant difference between groups (chi-square = 22.400, df = 2, $p < 0.0001$).

When the participants are considered as two groups only, control and diseased, i.e. presence of PTA regardless of clinical status, there was a significant difference in VISA-P between the two groups (Mann Whitney $p < 0.001$).

4.4 Body Mass

Descriptive statistics for body mass of the participants in each experimental group are provided in chart 4.2.

Chart 4.2: Body mass according to disease group



Box plot illustrates median (weighted horizontal line), interquartile range (box), extremes of data (whiskers) and outliers (dots).

A Kruskal-Wallis test showed that there was no significant difference between groups (chi-square = 2.468, df = 2, p = 0.291). When the participants are considered as two groups only, control and diseased, i.e. presence of PTA regardless of clinical status, the following results are obtained (table 4.1). There was no significant difference in mass between the control and diseased groups (p = 0.129).

Table 4.1: Comparison of body mass between control and diseased groups

	Control group (n=30)	**Diseased group (n=16)**	**Mann-Whitney *U* Test**	
	Median (IQR)	Median (IQR)	*U*	*p*-value (1-tailed)
Mass (kg)	93.442 (19.876)	100.270 (21.261)	191.00	0.129

IQR, interquartile range; U, Mann-Whitney U statistic

4.5 Playing position

The distribution of results according to clinical status separated by playing position is shown in chart 4.3 for forwards, and chart 4.4 for backs.

Chart 4.3: Distribution of results in forwards

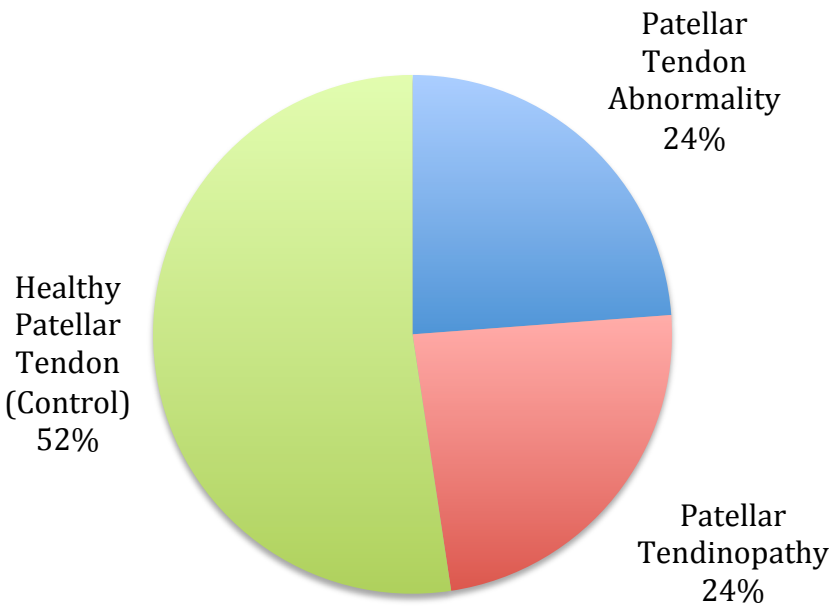
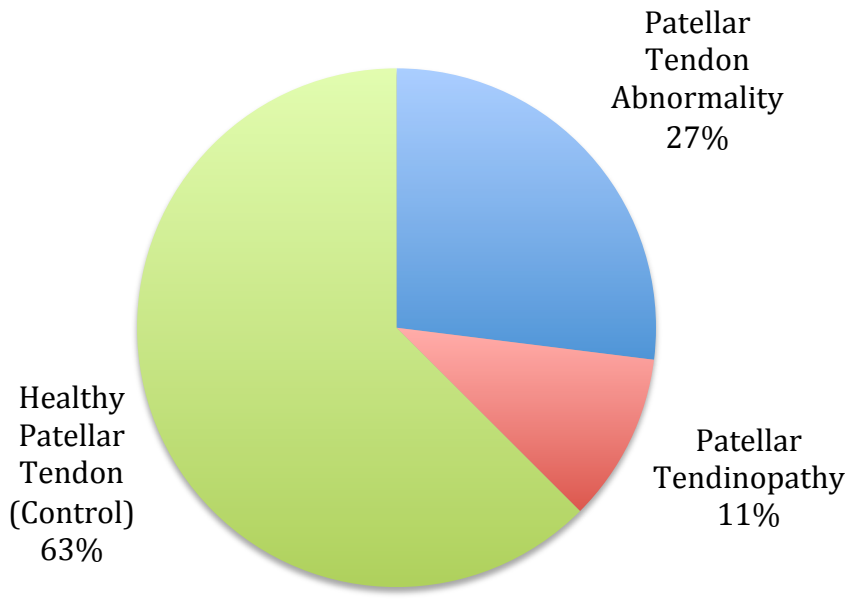


Chart 4.4: Distribution of results in backs

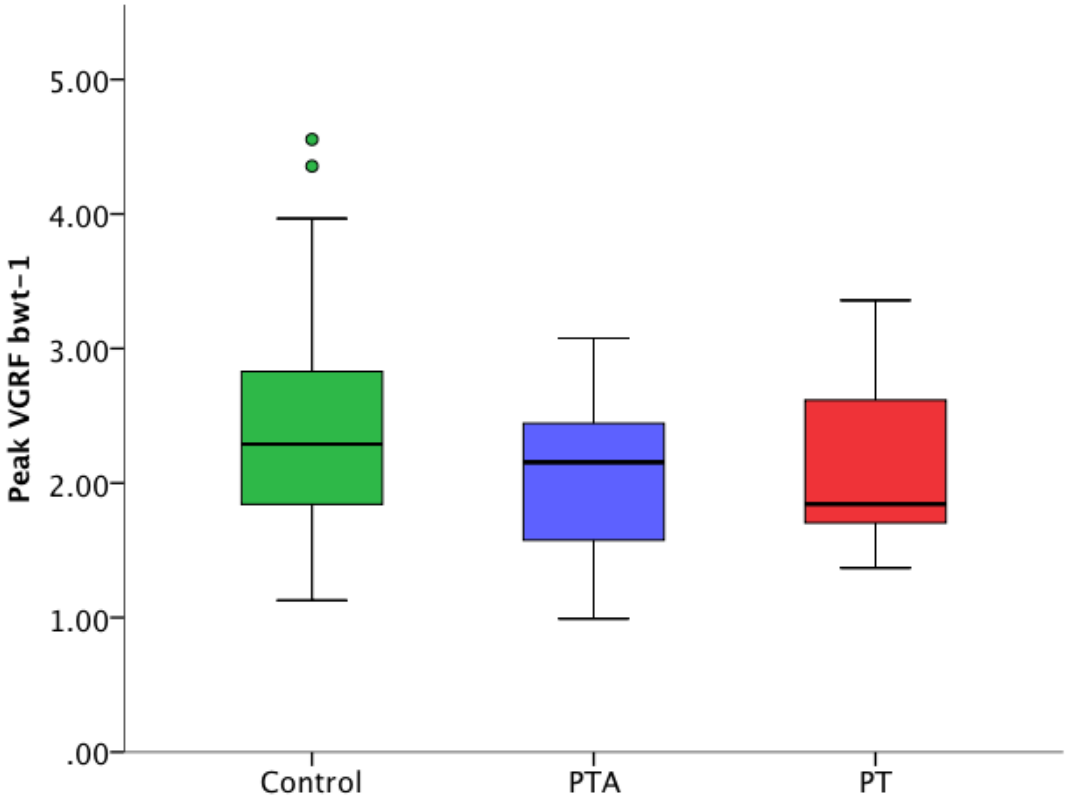


There was no significant difference in prevalence of PT or PTA in either forwards or backs ($\chi^2(2, N=46)=3.207$, p=0.201). The prevalence does not change when the participants are considered in two groups only, control and diseased i.e. presence of PTA regardless of clinical status ($\chi^2(1, N=46) = 2.807$, p=0.094).

4.6 Vertical Ground Reaction Force (VGRF)

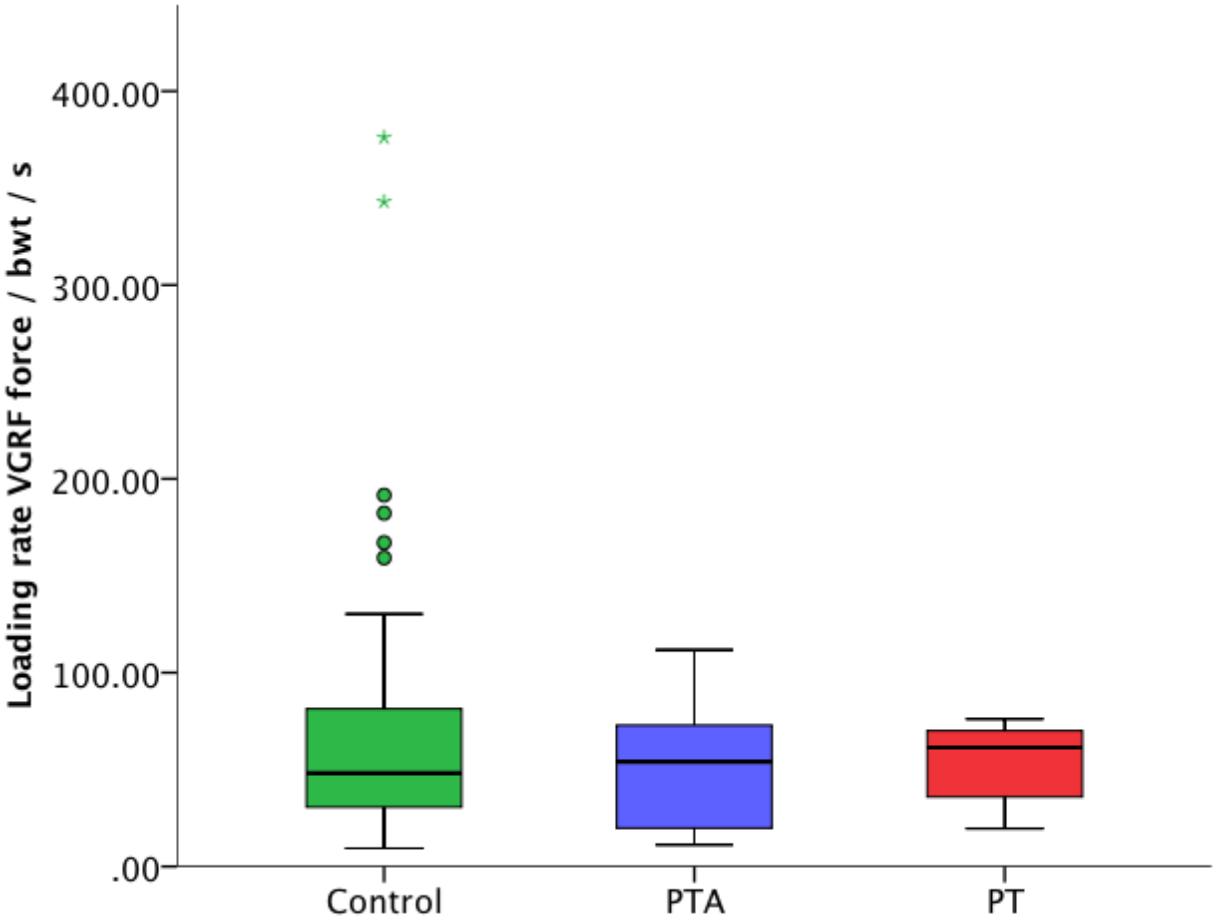
Descriptive statistics for peak VGRF and LR VGRF for tendons are provided in chart 4.5 and 4.6 respectively.

Chart 4.5: Peak VGRF according to disease group



Box plot illustrates median (weighted horizontal line), interquartile range (box), extremes of data (whiskers) and outliers (dots).

Chart 4.6: LR VGRF according to disease group



Box plot illustrates median (weighted horizontal line), interquartile range (box), extremes of data (whiskers), outliers (dots) and extreme outliers (asterisks).

A Kruskal-Wallis test showed that there was no significant difference between Controls, PTA and PT tendons for VGRF ($\chi^2 = 2.29$, df = 2, p = 0.318) or LR VGRF ($\chi^2 = 0.110$, df = 2, p = 0.946).

When the tendons are considered in two groups only, control and diseased (the presence of a PTA regardless of clinical status), the following results are obtained (table 4.2):

Table 4.2: Descriptive statistics for VGRF and LR VGRF

	Control tendons (n=71)	**Diseased tendons (n=21)**
	Median (IQR)	Median (IQR)
VGRF (bwt-^{1})	2.289 (1.037)	2.120 (0.931)
LR VGRF (force bwt-^{1} sec-^{1})	48.172 (54.733)	55.066 (49.750)

VGRF, peak vertical ground reaction force; LR VGRF, loading rate vertical ground reaction force; IQR, interquartile range.

The results above, suggest that it is unlikely that there is a difference between the diseased group and control in terms of force plate data (VGRF and LR VGRF). The smaller than desired sample size meant that it might in any case be difficult to detect such differences if they exist. However, as one of the key aims of the study was to assess whether ground reaction forces could be a predictor of patellar tendon disease, a regression analysis was undertaken.

A logistic regression model was conducted to test whether VGRF predicted clinical tendon status i.e. whether a tendon was healthy or diseased (presence of a PTA regardless of clinical status). Lower peak VGRF was not significantly associated with diseased tendons ($\chi^2 = 3.080$, $p = 0.079$; table 3). This was also true for LR VGRF ($\chi^2 = 1.567$, $p = 0.211$; table 5.3) i.e. lower LR VGRF was not associated with diseased tendons.

Table 4.3: Logistic regression model predicting likelihood to suffer diseased patellar tendon

	B	*p*	Odds ratio	95% confidence interval for odds ratio	
				Lower	Upper
VGRF	-0.614	0.097	0.541	0.262	1.118
LR VGRF	-0.007	0.276	0.993	0.981	1.006

VGRF, peak vertical ground reaction force; LR VGRF, loading rate vertical ground reaction force.

Data were obtained for each section of the study design as set out in chapter 2. The discussion of these findings is set out in chapter 5.

Chapter 5: Discussion

5.1 Introduction

In chapter 4 the results were presented that were used to investigate the objectives of the study. In this chapter, the results obtained have been discussed within the context of appropriate literature.

5.2 Sample

The response rate was 46 participants from a total possible sample of 80. When players were contacted directly via letter (as set out in the ethics submission), there were very few responses, which was very disappointing. Following this, contact was made with the regional physiotherapists. They were asked to approach players to request their participation and to hand them a copy of the letter, which set out the details of the study. The physiotherapists were asked to only approach potential participants who met the inclusion criteria. For example, they did not ask players who were currently injured. From this, 46 players consented to be involved. Clearly, involving the physiotherapists proved useful and such an approach would be recommended in future studies. Non-response was due mainly to unavailability or injury. The physiotherapists did not report that any player declined to be involved (apart from reasons concerning unavailability). In future, any power calculation would have to include non-response for these reasons in sample calculations.

5.3 Prevalence

As stated in the literature review, almost no studies report prevalence of patellar tendon disease in rugby players. However, there are studies in which PT in sports

that involve similar skills to rugby has been investigated (Ferretti et al., 1984; Ferretti et al., 1990; Lian et al., 2005). In this (Giles) study, six players (13%) from the sample reported a previous diagnosis of jumper's knee or patellar tendinopathy. Following discussions with squad physiotherapists since the data were collected, it appears that this 13% may be an underestimation. This is because it appears that physiotherapists do not always provide the player with a label such as "jumper's knee" or "patellar tendinopathy". In some instances, they merely treat and manage the symptoms. In this (Giles) study, it might have been more informative to ask questions regarding symptoms of patellar tendinopathy suffered previously rather than ask about a diagnosis having been given previously.

For those studies that do report prevalence of patellar tendinopathy in rugby (Brooks et al., 2005a, Brooks et al., 2005b, Fuller et al., 2008 and Fuller et al., 2012), there are issues around data collection (coding) and definition (acute versus chronic conditions), as has already been discussed. Since this study began, Durcan et al. (2013) examined the prevalence of patellar tendinopathy in elite academy rugby players in Ireland using almost identical data collection tools to the ones used in this study, which included clinical examination, ultrasound examination and VISA-P questionnaires. Their sample size was 83 players. It is not possible to be absolutely certain whether injured players were included in the study, which may explain their very high response rate (95.4%). The sample in Durcan et al. was very similar to the one in the present (Giles) study (see table 5.1).

Table 5.1: Comparison of demographic information with Durcan et al. (2013)

	Durcan et al. (2013) (n=83)	**Giles study (n=46)**
	Mean (S.D.)	**Mean (S.D.)**
Mass (kg)	97.6 (11.8)	94.3 (11.5)
Height (cm)	184.9 (7.1)	183 (5.97)
VISA-P score	92.1 (14.5)	92.9 (14.5)

It can be seen that the overall prevalence of disease was similar in the two studies (table 5.2). The prevalence of clinically apparent patellar tendinopathy in this (Giles) study (15.2%) is in line with PT estimations in the general elite sporting population i.e. 14% (Lian et al., 2005). Durcan et al. (2013) report more asymptomatic patellar tendon abnormalities (26.5%) than was the case in this study (19.6%). It is worth noting however, that Durcan et al. use very similar, but not identical, ultrasound criteria for diagnosing patellar tendon abnormalities. Nevertheless, both the figure for asymptomatic PTA found by Durcan et al. and this (Giles) study is similar to that reported for the presence of asymptomatic PTA by other studies i.e. 22-32% (Lian et al., 1996; Cook et al., 2000, Fredberg & Bolvig, 2002; Gisslèn et al., 2005; Malliaras et al., 2006).

Table 5.2: Comparison of results with Durcan et al. (2013)

	Durcan et al. (2013) (n=83)	**Giles study (n=46)**
Percentage of players with patellar tendon abnormalities on ultrasound	36.1	34.5
Percentage of players with patellar tendon abnormalities on ultrasound and with clinical symptoms and signs	9.6	15.2

Four players (8.7%) had bilateral disease according to US criteria in this (Giles) study. Again, this is similar to that reported by Durcan et al (9.7%). It is not possible to compare this with other studies because they either don't report it or report only in terms of total number of tendons. Durcan et al. report that the percentage of tendons that had an abnormal ultrasound appearance was 22.9%, compared with 21.8% in this (Giles) study. This makes the findings in both studies similar.

Both this (Giles) study and the Durcan et al. study involve academy rugby players and report a prevalence of patellar tendon disease at approximately 35%. This gives some reassurance that the findings in this (Giles) study are a reflection of reality. Nevertheless, the difference in prevalence in the two studies involving academy players from that of studies reporting prevalence in senior squads is substantial. For example, Brooks et al. (2005a, 2005b) report only three incidences of PT in 546 senior rugby players over two years. The reasons for this need to be explored but this may be because of two explanations.

The first explanation for the difference in reported prevalence may be that there is no real difference but the scale of the problem had been underestimated in previous studies because of problems with injury definition and coding, the diagnosis of patellar tendon disease and in the reporting of the condition. It is likely that under-reporting has resulted in such low prevalence rates because studies investigating prevalence in senior players have used injury self-reporting. The problems of self-reporting in chronic injuries, such as patellar tendinopathy, have already been discussed but include the fear of being diagnosed as injured and its implications (not playing, salary, career ending etc.). Perhaps, most seriously of all, given the findings in this (Giles) study and that of Durcan et al. (2013), the self-reporting approach to injury management and prevention is one which is not pro-active, i.e. players have to be sufficiently symptomatic before a diagnosis is attempted and thus reported. As many athletes are able to continue their sporting activities despite the chronic pain of patellar tendinopathy, they are often not included in incidence rates as new injuries that cause time-loss from sport (Scott et al., 2013). A more appropriate method for investigating overuse injuries has been recommended by Bahr et al. (2009) and advocated in the consensus statement from the International Scientific Tendinopathy Symposium in 2012 (Scott et al., 2013). Bahr made two main recommendations. The first is that "prevalence and not incidence should be used to report injury risk" (as was discussed in chapter 2). This is because incidence is an inappropriate measure for a condition that is both chronic and has insidious onset. The second recommendation is that "severity should be measured based on functional level and not time loss from sports" because athletes may be able to carry on playing and training despite the pain of overuse injuries. The VISA-P would be an example of a measure based on functional level in those with patellar tendinopathy.

The second explanation for the difference in reported prevalence of patellar tendon disease in rugby is that there is a true difference between the two populations studied (academy rugby players and senior rugby players). This may be due to age or training practices. On entering the academy system, age grade players are exposed to intensive training regimes, which exposes the athlete to increased load. Such a sudden increase in load (e.g. increased volume, intensity or frequency of training), which occurs when athletes are promoted from junior to senior levels, has been suggested as a possible risk factor for PT (Reeser et al., 2006 [citing personal communication from L Oystein and R Bahr 2006]. This supports the maxim that "tendons don't like rest or change" (Jill Cook, International Scientific Tendinopathy Symposium workshop, 2012). After discussion with the club physiotherapists involved in this thesis, it transpired that academy athletes might have more training and playing commitments than senior players. For example, a regional academy player may also be playing for (and training with) a local team and a university team. It follows that the player may be exposed to a load that is not fully appreciated by the regional physiotherapists, coaches, conditioners etc. Since tendon overload has been implicated as a critical factor in the development of patellar tendon disease, this needs to be given due consideration. Unfortunately, this (Giles) study was not designed to look at the difference in training practices between academy players and senior players. Despite this, it was worth considering whether the difference in prevalence in our (Giles) study, compared to epidemiological studies of senior players, may be due to the age and / or training practises of the athletes involved.

There was an opportunity to investigate further the cause for the difference in the reported prevalence. This is because the clinician who performed the ultrasound scans for this (Giles) study is also the physician to a Welsh regional rugby club. It is his normal practice to perform ultrasound scans on the squad. Routine ultrasound scans were performed on 35 senior players. The ultrasound scans were performed using the same equipment, the same methods and the same diagnostic criteria as used in this (Giles) study. Data were collected for 35 players and checked for accuracy. Permission was sought to release anonymised, aggregated data from the ultrasound scans so that comparisons could be made with this (Giles) data. The clinician's

normal practice to ensure that newly discovered injuries were dealt with was undertaken.

The results that follow help to inform the hypothesis that the difference in prevalence between academy players and senior players is due to different training regimes. However, it must be emphasized that this adjunct study was not part of the study design for this thesis. Informative though the results are, there needs to be a formally designed study that investigates the prevalence of PT in senior players, and compares this with the prevalence in academy rugby players. The results are summarised in table 5.3.

Table 5.3: Comparison of results with senior regional cohort

	Senior regional cohort (n=35)	**Giles study (n=46)**
Percentage of players with previous diagnosis of patellar tendinopathy	20.0	13.0
Percentage of players with patellar tendon abnormalities on ultrasound	22.9	34.5
Percentage of players with patellar tendon abnormalities on ultrasound, in the absence of clinical signs or symptoms	17.1	19.6

The results from the senior squad are similar to the academy players. There is no significance difference in the prevalence of patellar tendon disease between the two groups ($\chi^2(1, N=81) = 1.356$, $p=0.2443$). Therefore, this suggests that the prevalence of patellar tendon disease in senior rugby athletes has been previously underestimated. This is likely due to the problem with injury definition and coding (as has already been discussed). It gives further weight to the argument for using

alternative methods to quantify patellar tendon disease (and other overuse injuries) by using prevalence in prospective studies and measuring loss of function.

Similar to Durcan et al. (2013), there was a statistical significant difference between VISA-P score for control and diseased groups. These results should be interpreted with caution in terms of using VISA-P as a screening tool for patellar tendon disease. If the VISA-P was used for screening purposes, the symptoms reported on the questionnaire might not necessarily be attributed to PT. Therefore it lacks specificity. For example, the symptoms may be caused by other pathology (arthritis, recent trauma etc.). In addition, as is discussed in chapter 2, there may be a problem of under-reporting, which would result in false negatives. Furthermore, in this thesis, the VISA-P scores in the diseased groups have high standard deviations (11.4 and 22.4). This may be due to the subjective nature in the way which symptoms are experienced. This could also be a factor that undermines the utility of the VISA-P tool for screening purposes.

As documented in the section on ethical considerations, each player was informed of the findings of their clinical examination and ultrasound scan. Where abnormalities were present, the participants were informed that it would be beneficial to them that the findings were shared with their regional physiotherapist so that appropriate clinical management could be undertaken. Each player concerned consented to this. The relevant information was provided to the physiotherapist.

5.4 Body mass

The presence of disease was cross-tabulated with body mass. It was considered that there could be a relationship between body mass and the presence or absence of disease.

There was no relationship between body mass and presence of disease ($p=0.129$). This finding resembles that from Durcan et al. (2013) who also found no relationship between the two variables ($p=0.337$). In contrast, studies of patellar tendinopathy in

athletes from other sports, e.g. volleyball, had different results (Lian et al., 2003, $p=0.05$; Malliaras et al., 2007, $p<0.01$). Interestingly, the sample size in Lian et al. ($n=47$) is very similar to that of this study ($n=46$). However, the authors diagnosed patellar tendon disease via clinical methods only (with no reference to who did the examinations) and did not use musculoskeletal imaging. The authors did not identify players with patellar tendon disease based on ultrasound criteria and the results can therefore not be reliably compared with those in this study. Malliaras et al. (2007) defined patellar tendons as abnormal according to similar criteria as this (Giles) study, and from a sample of 73 males reported that weight was associated with abnormal imaging of the patellar tendon ($p<0.01$). This is counter-intuitive because the players in this (Giles) study and Durcan et al. (2013) had body mass greater than those in the Malliaras et al. (2007) study. This means that a factor other than body mass per se might have been responsible for the Malliaras et al. finding. This may be the result of the specific characteristics of each sport i.e. both sports involve eccentric loading, however volleyball is characterized by high frequency of maximal jump activities whereas rugby is characterized by a lower frequency of maximal jump activities. In addition, the different surfaces on which the two sports are played are likely to place different demands on the extensor mechanism of the knee. For example, Lian et al. (2005) cite Bahr and Reeser (2003) who showed that the prevalence in jumpers knee among elite beach volleyball players was only 9% which is considerably lower than for indoor volleyball players. They argued that the difference in prevalence of PT between the two sports when played on different surfaces is that jumping and landing in soft sand are less demanding on the patellar tendon than is jumping on indoor playing surfaces. Whereas indoor playing surfaces are designed to reduce the occurrence of overuse injuries by infrastructure that includes an element of shock absorption nevertheless, the playing surface is non-compliant wood. The rugby players in this (Giles) study played their competitive games on grass. Furthermore, personal communication with the regional academy physiotherapists revealed that the players in this (Giles) study had over 90% of their training sessions on grass. This could explain the difference in the prevalence of PT found in this thesis compared to those studying indoor volleyball but this hypothesis would need to be investigated in future studies.

5.5 Playing position

One of the risk factors identified for developing PT was position played with the proposition that forwards would be more at risk than backs. This is because traditionally forwards have greater body mass than backs, and are more likely to engage in maximal jump activities. In this (Giles) study, the results demonstrate no relationship between playing position and presence of patellar tendon disease ($p=0.094$). The only study that can be made reference to in relation to this finding is one by Lian et al. (2003), who found a statistically significant difference in the prevalence of patellar tendinopathy according to playing position in male volleyball players. However, it has already been discussed that the authors defined patellar tendinopathy without the use of ultrasound imaging and therefore it is difficult to compare the two samples.

5.6 Vertical Ground Reaction Force

Given that there is some evidence that athletes with patellar tendon disease exhibit different VGRF during landing tasks compared to their healthy counterparts, together with the speculation by Richards et al. (2002) that this easy to perform test may be used as a predictor of the presence or absence of disease, the relationship between VGRF and tendon status was examined.

There was no difference in peak VGRF or LR VGRF between controls and those with diseased patellar tendons (regardless of whether there were clinical signs or not). The logistic regression analysis was unable to predict tendon status in relation to ground reaction forces from drop-landings. These findings are counter to initial expectations. Once again, not reaching statistical significance might be the consequence of the small sample size. Initially, this was considered to be surprising because Fietzer et al. (2012) in a sample of only 18 elite dancers, found a significant difference in the peak VGRF between those with clinically diagnosed PT and asymptomatic controls ($p<0.001$). Further analysis of this paper revealed that it was likely that non-standardisation of both athlete and technique were factors at play,

which predisposed to statistically significant results being obtained. For example, males and females were included in the sample. In addition, participants were recruited from at least five different dance forms e.g. jazz, ballet etc. Also, the landing technique was not standardised. Participants were instructed to jump at full performance level, the height of which would have been different for each participant. The rugby participants in this (Giles) study all undertook a drop landing from an identical height (30 cm) to ensure that any difference detected in VGRF and LR VGRF would have mostly likely been to the presence of disease and not due to the height from which the landings occurred.

Bisseling et al. (2007) measured VGRF in a drop-jump protocol in their study into patellar tendinopathy in volleyball athletes. They reported no difference between athletes with patellar tendinopathy in peak VGRF but found a strong tendency ($p=0.05$) in higher LR VGRF when compared with controls. Again, the authors use a clinical diagnosis of PT to decide group membership. As such, the difficulties of comparing these results with this (Giles) study have already been discussed. The authors used a cross-sectional retrospective study design. Out of the 89 male volleyball athletes who completed the initial questionnaire, only 24 players were invited to take any further part in the study. Furthermore, group allocation (control, previous jumper's knee or recent jumper's knee) may have been influenced by recall bias. Coupled with the slightly different landing task involved, may explain the difference in the result regarding LR VGRF.

Edwards et al. (2010) used a stop-jump task to measure VGRF in controls and athletes with a PTA present on ultrasound imaging (but who are asymptomatic). There is no reference to the sports that these athletes play regularly apart from stating that their sports involve "repetitive landing". There could potentially be a wide range in the skills that these athletes possess. The vertical landing from a stop-jump task is not standardised unlike a drop-landing task. The vertical landing phase of the stop-jump task took place from a height that was, on average, higher than the 30 cm standardised height in the Giles study (50.5 +/- 5.9 cm for controls and 52.0 +/- 6.0 cm for PTA in Edwards et al.). The relatively small sample size (n=14, high standard deviations), and potentially involving athletes from a range of different sports mean

that the results must be interpreted with caution and cannot be directly compared with this (Giles) study.

However, the height of the drop-landing task (30 cm) in this (Giles) study might have been too low to detect any difference in VGRF due to patellar tendon disease. Other researchers have used greater heights from which to measure VGRF from landing tasks e.g. 50 cm and 70 cm, but these studies did not include rugby athletes. This issue was carefully considered (chapter 2) and the height that was thought to pose the least risk to players was chosen i.e. 30 cm. Following the drop-landing tasks, none of the participants reported pain or discomfort with the procedure. The game of rugby involves landing from heights greater than 30 cm. For future studies, the desirability of using greater heights for drop-landing tasks should be considered. It is premature to discount force plates and VGRF from having a role in screening for patellar tendon disease in athletes.

Conclusion

Given the current literature and the study objectives, it was surprising that no relationship was found between patellar tendon disease and key variables. This may be due to a smaller than desired sample size. However, if there is no relationship between the investigated key variables and patellar tendon disease, and this is considered alongside the high prevalence of the disease (validated by at least one other study), then all players must be considered to be at risk of developing the condition. It could also be the case that another variable, not yet considered, is the cause of high prevalence of patellar tendinopathy in rugby populations; e.g. load (high intensity, volume or frequency of training regimes). This should be considered for future study.

The prevalence rate of PT in this study and that of Durcan et al. (2013) is 15.2 and 9.6 % respectively. This is a high prevalence rate. However, it is based only on those athletes who were included in each study. For valid and meaningful prevalence rates, it is necessary that the entire *at risk* population be known accurately. The population

at risk is a group of people who share a characteristic that causes each member to be susceptible to a particular condition – or the number of people capable of experiencing the outcome of interest, i.e. patellar tendinopathy. With current data, it is impossible to be confident that the prevalence rates reported can be extrapolated to the population as a whole. That population might include all elite (or sub-elite) rugby players that may have to be divided into senior and youth athletes. The reason for caution is that prevalence rates are the most useful statistic both to quantify the size of the problem in a population and to plan services including disease management and prevention. As Chalmers (2002) argued, over a decade ago, we need to move from the maxim that "*injury is just part of the game*", to the maxim, "*injury prevention is just part of the game*". To realise this ambition, further studies to increase confidence levels in the prevalence rates discovered so far are required.

Chapter 6: Conclusions

This section on conclusions is derived from findings from both the literature review and the empirical work undertaken. This study of a sample of rugby athletes has provided insights into patellar tendinopathy: its prevalence, definition and the issues with coding. Hitherto weaknesses in all three may have led to an underestimation of the size and scope of the condition. To date, the majority of epidemiological studies into rugby report injury incidence rates and not prevalence. Often, chronic, overuse injuries, which have an insidious onset; of which patellar tendinopathy is one example; are not included in incidence rates as "new injuries" and this probably has resulted in under-reporting. Current methods to define injury, such as time-injury loss models, are not appropriate for patellar tendinopathy (or other overuse injuries). Rather, prospective models recording pain or function loss as a result of injury should be used. In addition, it is also essential that injury coding, which would inform prevalence studies, is valid. For example, injury codes should be specific and consistently used. This would overcome the problem of the vague way in which tendinopathy results have been presented in the literature which range from "knee injuries" to "tendon rupture / tendinopathy / bursitis" where the joint affected may or may not be specified.

The findings suggest that patellar tendinopathy is a significant problem in rugby union, with 15% of academy rugby players suffering clinically apparent patellar tendinopathy. A further 20% (35% total) of academy rugby players show patellar tendon abnormalities on ultrasound screening. Fortuitously, a contemporaneous study (Durcan et al., 2013) investigating patellar tendinopathy in rugby players has reported similar results. Both this (Giles) study and Durcan et al. report a higher prevalence than previously reported in the rugby literature, but consistent with the level of prevalence in sports that have been traditionally associated with the condition and which share similar characteristics to rugby i.e. jumping, sprinting and cutting. Thus, it has provided data regarding the prevalence of the disease, the first step in sports injury prevention models.

It was hypothesized that there would be a relationship between patellar tendinopathy and key variables (mass, playing position or VGRF from a drop-landing task). No statistically significant relationship between these variables was found. There are several possible explanations for this:

1. The small sample size. Several results were very close to being statistically significant and it might be that in a larger sample statistically significant results would be obtained. Given the high prevalence of disease, it is another reason for repeating the study with a bigger sample.
2. It may be that the height of the drop-landing task was inadequate to detect true differences in VGRF between players with healthy tendons and diseased tendons. Consideration should be given to repeating the study using a drop-landing task from an increased height i.e. 50 cm and 70 cm.
3. That there is no relationship could mean that all players, regardless of variables such as playing position, weight etc. must be considered to be at risk of developing PT. This may be due to an unknown factor e.g. load / training. The implications of this need careful consideration.

Despite the smaller than desirable sample size, a high prevalence rate of disease (one in three) was found and this was not related to mass or playing position. This finding was mirrored in a study carried out simultaneously in Ireland. Until more sophisticated prevalence studies can be conducted, it is reasonable to conclude that all players carry a risk of developing patellar tendon disease and further work to ensure that the population at risk is accurately captured is needed in order to provide a valid basis from which to perform future prevalence studies. There is a suggestion (from an informal, small set of data) that the prevalence of patellar tendinopathy in senior players resembles that found in the academy players that were included in this study. Given the career limiting possibilities of the condition, this finding has implications for the desirability for appropriate screening and research into risk factors and further studies are warranted.

This study should be repeated with a larger sample size because several of the results obtained approached statistical significance. It may well be possible to demonstrate

statistical significance with a larger sample, which would obviously add confidence to the findings.

Other conclusions in relation to sample are as follows:

1. Recruitment to the study proved very difficult until the cooperation of regional physiotherapists was obtained. Tactics such as this need to be adopted in future because one of the biggest causes of bias in research is a low response rate.

2. The sample size would have been bigger if the force plate equipment had not broken on the day scheduled to assess a further 23 players. In future, it is recommended that access to back up equipment, where possible, is essential.

3. There were no studies into patellar tendinopathy in rugby prior to the studies undertaken by Giles and Durcan et al. (2013), which occurred simultaneously. As a consequence of the results reported, future studies will be able to calculate power calculations to inform sample size requirements.

Limitations

The main limitations of this study relate to the smaller than desired sample size that was achieved. As has already been mentioned, due to the absence of pre-existing data regarding patellar tendinopathy in rugby players, it was difficult to undertake a power analysis prior to commencing the study. If possible, this would have given valuable information to suggest the sample size required to detect differences between those with patellar tendon disease and those without patellar tendon disease. As this was not possible, the protocols employed to detect true differences between those players with and without patellar tendon disease may have been compromised.

Study participants were required to provide information on whether they had previously suffered with jumper's knee or patellar tendinopathy. This may have been a source of recall bias and led to a misestimation of the scale of patellar tendinopathy. For example, a player may have been treated for patellar tendinopathy previously but had not been made aware of the diagnosis. One way in which this could have been avoided would have been to request medical notes from the players' medical team, however this would rely heavily on adequate medical notes being made contemporaneously. Adopting this approach would lead to ethical issues that would need careful consideration. However, since the examining clinician did not have access to this data (regarding previous diagnosis), this potential source of recall bias would not have influenced group membership i.e. whether a player was categorised as having patellar tendon disease or not.

Recommendations for practice and research

Notwithstanding these limitations, the results presented in chapter 4 give rise to several recommendations for clinical use and for areas of future research. The recommendations for clinical practice and sports injury prevention are as follows:

1. Health professionals looking after rugby players (particularly age-grade players as they progress through the academy system) should be aware of this high prevalence and be actively looking for symptoms and signs of patellar tendinopathy. Players should be instructed as to the symptoms of patellar tendinopathy and encouraged to report such symptoms to their medical team or physiotherapist.

2. All elite rugby players, particularly age-grade players, should have their load monitored and managed appropriately.

3. Due to the high prevalence of asymptomatic PTA found in this study, coupled with the higher risk of athletes with PTA going on to develop symptoms of patellar tendinopathy, it is desirable to screen all elite rugby

players. Given that this study (with its smaller than desired sample size) does not support the use of VGRF (from drop-landing tasks at heights of 30 cm) in the screening for patellar tendon disease, and in the absence of an alternative method at this time, the method of screening for patellar tendon disease should be ultrasound screening. Despite the difficulties associated with this recommendation (particularly the expense and expertise required) a balance has to be struck between health of the athlete and expense. In order to support the medical team, recommendations for ultrasound screening to be made available should be made to rugby governing bodies.

The recommendations for future research are as follows:

4. The prevalence of patellar tendon disease in rugby has been found to be higher than previously thought. To ensure that the population at risk is accurately captured, the study should be repeated with a larger sample size. This should include senior rugby players (i.e. not just those attached to academies). This would improve the external validity of the results. It would also clarify whether any differences in prevalence exist for both age-grade and senior athletes. For future studies, it will be possible for power calculations to determine the required sample size, because this (Giles) study has provided data on mean and standard deviation for key variables.

5. Future studies should consider mechanical overload as a factor that contributes to the development of patellar tendon disease in rugby athletes. The definition of overload might be different for age-grade players and senior players. By collecting data on variables such as frequency, volume of training, playing intensity etc. the potential effects of overload can be studied appropriately. Notational analysis methods would facilitate such investigations.

6. When investigating prevalence of patellar tendinopathy, it would be favourable to ask players whether they had suffered with the symptoms of patellar tendinopathy previously rather than ask whether they had been given a diagnosis of the condition. Physiotherapists or clinicians may merely treat

the symptoms of patellar tendinopathy rather than explain the diagnosis to the player concerned. Adopting this approach (of enquiring about symptoms rather diagnosis), would likely result in a more accurate estimation of the lifetime prevalence of patellar tendinopathy.

7. As no participant, including those with patellar tendon disease, found the drop-landing task uncomfortable, future research investigating VGRF in rugby athletes should use a height of greater than 30 cm for the drop-landing task.

8. The findings to date, do not allow predictions to be made about which players with asymptomatic PTA go on to develop symptomatic patellar tendinopathy. It is recommended that the players identified as having an asymptomatic PTA in this study should be invited to participate in a follow up study to determine how many players with PTA (identified on ultrasound) went on to develop symptoms. It would then be possible to explore whether any relationship existed between those that went on to develop symptoms and their baseline VGRF data. Whilst being mindful that the numbers involved in this sample subset would be small, any insights would add strength to the recommendation to repeat the research with a larger sample.

9. It is not known whether other lower limb injuries predispose athletes to developing patellar tendinopathy. Studies often list previous injuries as an exclusion criterion as a previous injury may act as a confounding variable. Since injuries are becoming increasingly common among athletic populations, future studies should aim to investigate whether there is an association between previous injury and patellar tendinopathy.

10. As the prevalence of patellar tendon disease is high, and given the career limiting possibilities of the condition, there is a need for further high quality research into patellar tendon disease in rugby athletes with specific emphasis on consistency in diagnosis and coding. If this were achieved, it would enable reliable large data sets to be available for analysis.

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Appendix 1. Ethical approval

University of Glamorgan
Prifysgol Morgannwg
Faculty of Health, Sport and Science
Cyfadran Iechyd, Chwaraeon a Gwyddoniaeth

25 August 2011

Mr M Giles
C/o Faculty of Health, Sport and Science

Dear Mr Giles,

Re: The relationship between ground reaction force and patella tendinopathy

I am writing to confirm that on the 25 August 2011, the Faculty of Health, Sport, and Science Ethics Sub Group approved your revised low risk submission for ethical approval, via Chair's action.

If you have any queries about the group's decision, please do not hesitate to contact me. Please note: when changes are made to an already approved protocol the opinion of the Faculty Ethics Champion must be sought.

Yours sincerely,

Professor Paul Rogers
Faculty Ethics Champion

Appendix 2. Study invitation letter to regional rugby academies

Dear (manager / medical staff)

The University of Glamorgan is about to launch research into a new method of screening for patellar tendinopathy. It is one of the research topics sponsored by the JPR Williams trust.

It will investigate whether force plate testing would be a suitable method for screening for this common condition. This would hopefully allow the problem to be picked up before symptoms start and would avoid both impaired performance and time off with injury.

Force plate analysis is a simple test which can potentially be performed at club training grounds and would avoid the need for players to travel to a specialist medical centre unnecessarily.

The research will involve inviting players to undergo a force plate analysis, answering a questionnaire and having an ultrasound scan of their patellar tendons. The whole process should take no more than 40 minutes per player. Neither the force plate testing or ultrasound scan causes any discomfort.

Any player (with their consent) identified as having a potential problem with their patellar tendon will be referred to a sports medicine physician and/or club physiotherapist for further investigation and management.

All elite academy players in Wales will be offered to be involved in the research, and we would initially aim to visit yourselves and discuss the opportunity with both you and the eligible players. We will also use this time to answer any questions or concerns you may have.

Dr M Giles

JPR Williams Research Fellow
University of Glamorgan

Appendix 3. Participant Information Sheet

Participant Information Sheet

Project Title: *The relationship between ground reaction force and patellar tendinopathy*

You are being invited to take part in a research study. Before you decide whether or not to be involved, you will need to know why you are being asked to take part and what the process involves. Please take time to read the following information carefully. Please ask us if there is anything you are not sure about or if there's anything you would like to know more about.

What is the purpose of the study?

Patellar tendinopathy or *jumpers knee* is commonly seen in rugby players and can limit or even end a promising professional career. It is caused by repetitive movements such as jumping or sudden changes in direction while running. It results in pain felt over the lower part of the knee.

Patellar tendinopathy often causes the player to adapt the way they move, jump and run. Patellar tendinopathy increases the possibility of a potentially serious injury.

A diagnosis of jumpers knee is usually made by clinical examination by a doctor or physiotherapist. It is confirmed with an ultrasound scan of the knee. The changes on this ultrasound scan can often be seen before the athlete develops pain. However, ultrasound scanning requires a great deal of cost and expertise.

It may be that those athletes with degenerative patellar tendons have different forces exerted on them by the ground compared with athletes without these degenerative changes. This can be measured using a force plate.

The aim of this study is to investigate whether force plates may be used as a more accessible method than ultrasound in *screening* for signs of patellar tendinopathy. This would allow players to be screened and treated for this condition before they develop pain.

Why have I been chosen?

Patellar tendinopathy commonly affects rugby players as well as other athletes - more commonly around adolescence. As such, all age grade elite rugby players from the four regions in Wales will be invited to participate in this study.

Do I have to take part?

It is up to you whether or not to take part. There are no penalties to refusing to take part and you do not have to give a reason. If you agree to take part, you will be given this information sheet to keep and you will be asked to sign a consent form to show you agree to take part. You are still free to withdraw from the study at any time.

What will happen to me if I take part?

If you agree to take part you will be invited to attend the University of Glamorgan at a time convenient to you. This is the only visit you will make. This visit will be split into three stations.

Station 1: A short questionnaire (approx. 5 minutes)

This will involve giving details regarding your date of birth, whether you are left or right handed, preferred playing position and answering some questions regarding whether or not you experience knee pain.

Station 2: Examination and scan of your knees by a doctor (approx. 10 minutes)

This will involve feeling the lower part of your knee for pain and asking you to do some simple movements. This section will end by having an ultrasound scan of your knees, this is a painless procedure. There is no discomfort experienced with an ultrasound scan.

Station 3: Force plate analysis (approx. 15 minutes)

This will involve stepping off an elevated platform on to a force plate. This force plate will measure the force generated when your feet touch the ground. You will be asked to do this three times. No discomfort is experienced with this procedure.

What else do I need to do?

There are no restrictions to your lifestyle or changes in your training programme that you need to make in order to take part in this study.

What are the possible disadvantages and risks of taking part?

There are no foreseeable risks to taking part in this study.

What are the benefits of taking part?

This study may identify those athletes with patellar tendinopathy. If this happens, we will inform your club physiotherapist (with your permission) so that they can start treating you for the condition.

We cannot guarantee that taking part in this study will help you as an individual, but the information we get from the study may help to screen athletes for patellar tendinopathy in the future.

Expenses

All travel costs incurred will be refunded by the University of Glamorgan

Will my taking part in the study be kept confidential?

Yes, all information collected during the study will be kept strictly confidential. Each participant will be given a unique research code known only to the research team. Any data which is disseminated will have your identifiable information removed so that you cannot be traced.

What will happen to the results of the research project?

The results of the study will form part of a thesis. The results may also be submitted for publication in scientific journals and presented at scientific meetings. No participant will be identified in any publication of the results.

The data collected in this project may be used in subsequent or additional ethically approved projects but no individual shall be named. The results will be made available to your region and you will be invited to a seminar in which the results are to be presented.

Who is organising and funding the project?

The project is being organised by the University of Glamorgan and joint funded by the University of Glamorgan and JPR Williams Trust.

Who has reviewed the project?

This project has been approved by the University of Glamorgan local ethics committee.

Who should I contact for further information?

For further information on patellar tendinopathy, the following websites may be useful:

1. http://www.bupa.co.uk/running/injury-prevention-and-recovery/injuries/patellar-tendinopathy/
2. http://www.sportsinjuryclinic.net/cybertherapist/front/knee/indexjumpersknee.html

For specific information regarding this project, in the first instance you can contact

Dr Matthew Giles	email: matt.giles@me.com
Professor Gareth Jones (supervisor)	email: gareth.jones@doctors.org.uk
Morgan Williams (supervisor)	email: mdwillia@glam.ac.uk
Professor Richard Mullen (supervisor)	email: rhmullen@glam.ac.uk

If you have any concerns during the study or if there's anything you are unhappy about, you may contact the University of Glamorgan complaints procedure.

Appendix 4. Consent form

Project title: *The relationship between ground reaction force and patella tendinopathy*

I agree to take part in the above faculty of health, sports and science (University of Glamorgan) research project. I have had the project explained to me and I have read the participant information sheet.

I understand that agreeing to take parts means that I am willing to:

Answer a questionnaire giving information on general characteristics, whether I experience any symptoms of patellar tendinopathy and how (if applicable) they affect my quality of life
Receive an examination of the knee by a Sports Medicine doctor
Receive an ultrasound scan of the knee
Participate in force plate testing

Data protection

The information collected for the study will be held and processed only for the purposes of research.

I understand that any information I provide is confidential, and that no information that could lead to the identification of any individual will be disclosed in any reports on the project, or to any other party. No identifiable personal data will be published. The identifiable data will not be shared with any other organisation or individual.

I agree to the University of Glamorgan collecting and processing this data. I understand that the data will be used only for the purpose(s) set out in this statement and my consent is conditional on the University complying with its duties and obligations under the Data Protection Act 1998.

Withdrawal from study

I understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalised or disadvantaged in any way. Should I decide to withdraw from the study this decision will not be communicated to a third party.

I agree that the data collected in this project may be used in subsequent or additional ethically approved research projects. I understand that no identifiable personal information will be shared or published.

Name: ..... (please print)
Signature: .....……
Date: .....

Independent witness to participant's voluntary and informed consent:

I believe that …….. understands the above project and gives her/his consent voluntarily

Name: ..... (please print)
Signature .....
Date: .....
Address: .....

Appendix 5. VISA-P questionnaire

Faculty of Health, Sports and Science, University of Glamorgan

Victorian Institute of Sport Assessment (VISA-p) questionnaire

Points

1. For how many minutes can you sit pain free?

0 min ☐☐☐☐☐☐☐☐☐☐☐ 100 min

0 1 2 3 4 5 6 7 8 9 10

Points

2. Do you have pain walking downstairs with a normal gait cycle?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain

0 1 2 3 4 5 6 7 8 9 10

Points

3. Do you have pain at the knee with full active non-weight-bearing extension?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain

0 1 2 3 4 5 6 7 8 9 10

Points

4. Do you have pain when doing a full weight bearing lunge?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain

0 1 2 3 4 5 6 7 8 9 10

Points

5. Do you have problems squatting?

Unable ☐☐☐☐☐☐☐☐☐☐☐ No problem

0 1 2 3 4 5 6 7 8 9 10

Points

6. Do you have pain during or immediately after doing 10 single leg hops?

Strong severe pain/ unable ☐☐☐☐☐☐☐☐☐☐☐ No pain

0 1 2 3 4 5 6 7 8 9 10

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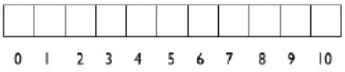
Victorian Institute of Sport Assessment (VISA-p) questionnaire

1. For how many minutes can you sit pain free? Points ☐

0 min  100 min

2. Do you have pain walking downstairs with a normal gait cycle? Points ☐

Strong severe pain  No pain

3. Do you have pain at the knee with full active non-weight-bearing extension? Points ☐

Strong severe pain  No pain

4. Do you have pain when doing a full weight bearing lunge? Points ☐

Strong severe pain  No pain

5. Do you have problems squatting? Points ☐

Unable  No problem

6. Do you have pain during or immediately after doing 10 single leg hops? Points ☐

Strong severe pain/ unable  No pain

Appendix 6. Experience of clinician conducting clinical examination and undertaking ultrasound scans of patellar tendons

Professor Gareth Jones

Qualifications

MB BCh	1976
MRCP (UK)	1984
MRCGP	1986
MSc (SEM)	2003

Membership and affiliations

FFSEM (I)	2004
MFSEM (UK)	2005
BASEM	

Relevant courses

MSc (SEM) Bath University 2003

Musculoskeletal Ultrasound Intervention course 2011

Musculoskeletal Ultrasound (Philips Medical systems) 2006

Current Practice and experience

Specialist doctor in cardiology (NHS) – one session per week

1998-present

General cardiology (including echocardiogram) and manages rapid access chest pain clinic for GPs and secondary care physicians within Hywel Dda university health board

Specialist doctor in in orthopaedics (NHS) – two sessions per week
2013-present
Provides clinical assessment, near patient musculoskeletal ultrasound and ultrasound guided injections. Routinely undergoes audit comparing pre-operative ultrasound reports with findings at operation
Undergoes 6 weekly peer review MSK-US with consultant radiologist

Cardiff Blues head sports physician – three sessions per week (and matches)

Provides cardiac screening (including echocardiograms) to WRU – national seniors, 7s, and national age-grade squads

Course director, author and tutor – online diploma / MSc in Sports & Exercise Medicine (University of South Wales)

Provides clinical expertise and support to SEM research studies at the University of South Wales

Bath University Tutor and examiner for Dip/MSc SEM

Provides sports medicine services to team sport at the University of South Wales

Chief Medical officer Gemau Cymru (2011-present)

Welsh Rugby Union Sports physician 1993-2013

Ospreys rugby sports physician (2003-2012)

Commonwealth Games council for wales (1998 – Team doctor, 2002-2010 – Chief medical officer)

Tumble RFC Team doctor (1987-1995)

Appendix 7. Pro-forma for data collection: clinical examination

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Assessment by Sports Physician

Pain over inferior pole of patella (consistent with PT)?

Left side:	Yes	☐	No	☐
Right Side:	Yes	☐	No	☐

Pain in palpation over patellar tendon (consistent with PT)?

Left side:	Yes	☐	No	☐
Right Side:	Yes	☐	No	☐

Clinically does this player signs consistent with patellar tendinopathy?

Left side:	Yes	☐	No	☐
Right Side:	Yes	☐	No	☐

Appendix 8. Pro-forma for data collection: ultrasound examination

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Ultrasound scan of patellar tendon

Left side: Tendon width (mm) .....

Right side: Tendon width (mm) .....

Any hypoechoic areas evident (on both views)?

Left side: Yes ☐ No ☐

Right Side: Yes ☐ No ☐

Inhomogeneity present?

Left side: Yes ☐ No ☐

Right Side: Yes ☐ No ☐

Evidence of neovascularisation?

Left side: Yes ☐ No ☐

Right Side: Yes ☐ No ☐

On ultrasound, does this person have a PTA?

Left side: Yes ☐ No ☐

Right Side: Yes ☐ No ☐